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A CLINICAL TEXT-

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PHYSICIANS AND S

BASED ON THE

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FRANCIS H. STUART

FOURTH AMERICAN EDITION, REVISED

THE LEHMAN PUBLISHING

A CLINICAL TEXT-BOOK

OF

MEDICAL DIAGNOSIS

FOR

PHYSICIANS AND STUDENTS

BASED ON THE MOST RECENT METHODS OF EXAMINATION

BY

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AUTHORIZED TRANSLATION

WITH ADDITIONS

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TRANSLATOR'S PREFACE TO THE FOURTH AMERICAN EDITION.

PROFESSOR VIERORDT'S "Diagnostik der Innerer Krankheiten" has created and met a demand which is remarkable in medical literature. Less than nine years elapsed between the date of the first edition and that of the fifth, from which the present translation is made. During this period the progress in various departments of diagnosis has been great, but the book has been kept fully abreast of this progress: the Author has carefully revised his work four times.

Upon its first publication it was immediately translated into Russian and Italian as well as into English, and was welcomed by the medical profession in all parts of the globe.

It will be found that all the qualities which made the earlier editions so acceptable have been developed with the evolution of the work to its present form. A distinguished professor of medicine who also has a large consultation practice once said to the translator, "I have never read a medical work from which I derived so much profit as from Vierordt." It is a veritable mine of information on all points in medical diagnosis.

It is therefore a great pleasure for the translator to present to the English-reading medical public this fourth edition. The labor for one in active practice has been great, but it is ample reward to have thus kept pace with the diligent author by bringing the work up to date in its English dress. What was first sent forth with fear lest the judgment of others would not be consonant with his own, is now given with assurance that it will meet with a still warmer welcome by reason of its enhanced and inherent excellence.

FRANCIS H. STUART.

123 JORALEMON STREET, BROOKLYN, NEW YORK, N. Y.,
August 1, 1898.

AUTHOR'S PREFACE TO THE FOURTH AMERICAN EDITION.

In the first place, it gives me pleasure emphatically to express my approbation of Dr. Francis H. Stuart's translation of my work upon Diagnosis as being accurate and excellent in every respect. I rejoice in its great success.

The present edition in English is translated from the fifth German edition, for which I furnished advance sheets. Many alterations have been made throughout the book, but especially in the sections on Gastric Digestion and the Nervous System.

The particular purpose of my work is to furnish the physician with the material by which he may make himself an accomplished diagnostician in all branches of medical diagnosis. The foundation of a correct diagnosis must rest upon a careful examination of the individual organs, and then a study of the whole organism, the totality of the picture of the disease.

May the new edition in English, to which I wish the same success the former editions had, contribute to the attainment of this high end.

PROF. O. VIERORDT.

Heidelberg, February 5, 1898.

PREFACE TO THE FIFTH GERMAN EDITION.

THE new edition has been revised in all its parts, and altered or enlarged in many places. The most thorough revision has been made in regard to the examination of gastric digestion and the examination of the nervous system. Fourteen new illustrations have been added, and a few former ones have been replaced by ones more suitable. For a part of these I have to thank my former assistant, Dr. Becker.

That we have for the present altogether omitted the application of Röntgen rays for the purposes of internal medicine, which is still in its first beginnings, will no doubt be approved by every reader.

Nevertheless, a not inconsiderable enlargement of the book could not be avoided. I hope that this enlargement will also be considered an improvement.

O. VIERORDT.

HEIDELBERG, FEBRUARY, 1897.

TRANSLATOR'S PREFACE.

THE work of which a translation is here offered is one of the best that has yet been written upon the subject. When it first came into the hands of the translator he had no thought of ever using it except as a work of reference. But as he read it he became convinced that it had such merit that it would certainly be welcomed by a large class of readers if it were rendered into English. Accordingly, after communicating with the author and his publisher, the work of translation was begun, and has been prosecuted at such intervals of time as could be secured from an active professional life. If the work shall commend itself to others as it has to him, the translator will feel amply rewarded for the effort he has made to put it into their hands.

Here and there slight additions have been made, which the translator trusts will increase the value of the work. A very full index has been prepared, which, it is believed, comprises a reference to every material statement in the book.

The translation was almost completed when a copy of the second edition of the original was received from the publisher. The author has made numerous additions which have enhanced its value, and the translation has been made to correspond with this enlarged edition. It is gratifying to the translator to find that a second edition has so soon been called for, and that his own favorable opinion has been further confirmed by the fact that Italian and Russian translations of the work have been made.

FRANCIS H. STUART.

123 JORALEMON STREET, BROOKLYN, N. Y.,
March, 1891.

PREFACE TO THE FIRST EDITION.

THE book which is here offered to the medical public was undertaken at the solicitation of a number of associates, and in view of the experience which I have acquired during more than four years of work as Teacher of Diagnosis in the Medical Clinic at the University of Leipsic. Originally I had in view a very extensive treatise comprising a detailed explanation of normal and pathological anatomy and physiology as a foundation for diagnosis. But this plan I abandoned with a view to the convenience and general usefulness of the book.

Regarding the principles which have guided me, and which I hope, particularly in the "Special Part," notwithstanding the brevity of the presentation, have been made plain, I may be permitted here to specify the following. I have here, as well as in my teaching, taken pains to emphasize that, besides availing ourselves of the constantly-increasing finer methods of diagnosis, the simple use of our senses, especially of the unaided eye, must not be forgotten. Still more, the manifold labors with the microscope and in the laboratory ought not to permit the physician to forget that a preparation or a chemical reaction is not enough for a diagnosis, but that the whole organism must always be brought under consideration. In other words, in diagnosis as well as therapeutics this rule is imperative: We must *individualize* the case. Should the book to any extent antagonize the inclination of our time to theorizing, it would afford me especial satisfaction.

OSWALD VIERORDT.

LEIPSIC, JUNE, 1888.

CONTENTS.

PART I.

CHAPTER I.

Introduction.

Anamnesis
Mode of Taking the Anamnesis
What the Anamnesis Comprises
Previous History of the Patient
The Present Disease

CHAPTER II.

Examination of Patients

What the Examination Comprises
Note by the Transcriber upon Keeping records of cases and
Results of a Medical Examination

PART II.

CHAPTER III.

General Examination

I. The Psychic Condition of the Patient
II. The Position of the Moment
III. The Structure of the Body and Extremities
IV. Skin and Subcutaneous Cellular Tissue
A. The State of Nutrition of the Skin
B. The Muscles and the Skin
C. The Glands of the Skin
1. The Hair Glands
2. Abnormal Secretions of the Skin
3. The Hair and Nails
4. The Sweat Glands
5. The Pigment Glands
6. The Cutaneous Sensibility

CONTENTS.

PART I.

CHAPTER I.

Introduction.

PAGE

Anamnesis 19
Mode of Taking the Anamnesis 19
What the Anamnesis Comprises 20
Previous History of the Patient 20
The Present Disease 22

CHAPTER II.

Examination of Patients.

What the Examination Comprises 24
Note by the Translator upon Keeping Records of Cases, and a Form for Recording the Results of a Medical Examination 24

PART II.

CHAPTER III.

General Examination.

I. The Psychical Condition of the Patient 28
II. The Position of the Patient 28
III. The Structure of the Body and Nutrition; Weight 30
IV. Skin and Subcutaneous Cellular Tissue 32
A. The State of Nutrition of the Skin 32
B. The Moisture of the Skin; Perspiration 32
C. The Color of the Skin 34
1. The Pale Skin 35
2. Abnormal Redness of the Skin 37
3. The Blue-red Skin, Cyanosis 38
4. The Yellow Skin, Icterus, Jaundice 40
5. The Bronze Skin 43
6. The Gray Skin of Silver Deposit 44

PAGE

D. Other Pathological Appearances of the Skin of General Diagnostic Value . . 44
1. Acute Exanthematous Diseases 44
2. Exanthemata from Poisons and the Use of Medicines 45
3. Hemorrhages in the Skin 45
4. Scars 46
E. Edema of the Skin and Subcutaneous Cellular Tissue (Edema, Anasarca) . . 46
F. Emphysema of the Skin 49
V. The Temperature of the Body; Fever 50
1. The Terms Used and the Method of Taking the Temperature 50
2. The Normal Temperature of the Body 53
3. Elevated Temperature; Fever 54
4. The Subnormal Temperature 56
5. Diagnostic Value of the Temperature, especially of its General Course . 57
6. Local Elevation or Lowering of the Temperature 65

PART III.

CHAPTER IV.

Examination of the Respiratory Apparatus.

Examination of the Nose and Larynx 66
1. The Nose 66
2. The Larynx 67
(*a*) Its Function 67
(*b*) The Internal Examination 68
Examination of the Lungs 68
Topographical Anatomy of the Lungs 68
The Anatomical Boundaries of the Lungs with Reference to the Thorax 69
Inspection of the Thorax 72
1. Normal Form of Thorax and Normal Respiration 72
2. Pathological Forms of Thorax 75
3. Anomalies of Respiration 79
(*a*) Anomalies of the Manner of Breathing 80
(*b*) Anomalies of the Frequency and Rhythm of Breathing 81
(*c*) Difficult Breathing, Dyspnea 83
Palpation of the Thorax 89
1. Pain caused by Pressure upon the Thorax 89
2. Testing the Movements during Respiration 91
Percussion of the Thorax 92
General and Preliminary Remarks Regarding Percussion 92
1. History and Methods 92
2. Qualities of Sounds 94
3. The Conditions that Determine the Quality of the Sounds and their Production in the Body. The Feeling of Resistance 96
4. Topographical Percussion: Determining the Parietal Boundaries of Organs 102
Percussion of the Thorax, Especially of the Lungs 103
1. Methods 103

PAGE

2. Normal Sound over the Lungs, Trachea, and Larynx. The Normal Boundaries of the Lungs 105
3. Abnormal Sound over the Lungs. Abnormal Position of the Border of the Lungs 109
A. Dulness: Deadened Resonance 109
B. Tympanitic Resonance 112
C. Abnormally Loud and Deep Sound 116
D. Changed Condition of the Boundaries of the Lungs 117
Auscultation of the Lungs 118
1. History. The Sphere of Auscultation at the Present Time 118
2. Methods of Auscultation 119
3. Auscultatory Signs in Normal Respiration 121
4. Pathological Sounds in the Respiratory Apparatus 123
(*a*) Alterations of Vesicular Breathing 124
(*b*) Bronchial Breathing 125
(*c*) Undefined Breathing 127
(*d*) Dry Râles (Rhonchus, Humming, Whistling, Hissing) 128
(*e*) Moist Râles 129
(*f*) Crepitant Râles (Crepitation) 131
(*g*) Pleuritic Friction Sounds 132
(*h*) Hippocratic Succussion 133
Palpation of Vocal Fremitus (Auscultation of the Voice) 133
Exploratory Puncture of the Pleura; Diagnostic Study of the Fluid Obtained by Puncturing 136
1. Mode of Procedure 136
2. Chemical Examination of the Aspirated Fluid 139
Methods of Measuring and Stethography 140
Measuring the Thorax 140
Spirometry, Pneumatometry, and Stethography 141
Cough and Expectoration 142
Expectoration, Sputum 143
1. General Characteristics of the Expectoration 144
2. Foreign Substances in the Sputum which are Visible to the Unaided Eye . 147
3. Microscopical Examination of the Sputum 150
4. Chemical Examination of Sputum 164

CHAPTER V.

Examination of the Circulatory Apparatus.

Examination of the Heart 166
Anatomy of the Normal Heart 166
Preliminary Remarks necessary to Understand the Physical Phenomena of the Heart 167
Inspection and Palpation of the Region of the Heart 171
The Apex-beat; Normal Conditions 171
Alteration in the Width and Strength of the Apex-beat 173
The Neighborhood of the Heart in General 175
The Epigastrium 177
Percussion of the Heart 177
Normal Percussion-figure of the Heart, Methods of Percussion 177
Enlargement of the Area of Heart-dulness 180
Diminution or Loss of Heart-dulness 181
Displacement (Dislocation) of the Heart-dulness 182

PAGE

Auscultation of the Heart 182
Methods and Normal Condition 182
Pathological Changes in the Heart-sounds 187
Organic Endocardial Heart-murmurs 191
Inorganic, Anemic Murmurs 197
Pericardial Murmurs. [Friction-sounds.] 198
Examination of the Arteries 201
I. The Pulse, its Palpation and Graphic Representation 201
Palpation of the Pulse 201
1. The Normal Pulse 201
2. Pathological Frequency of the Pulse 203
3. Want of Rhythm of the Pulse 207
4. Quality of the Pulse 208
5. Symmetry of the Radial Pulse 210
Sphygmography of the Radial Pulse 211
Diagnostic Value of the Examination of the Pulse 216
II. Other Phenomena in Arteries 218
The Aorta 218
The Pulmonary Artery 219
The Other Arteries 220
Examination of the Veins 223
Inspection and Palpation of Veins 223
1. Increased Fulness of Veins 223
2. Phenomena of Circulation in the Jugular Veins 225
3. Phenomena of Circulation in Other Veins 229
4. Venous Thrombosis 229
Auscultation of Veins 230
Examination of the Blood 231
Preliminary Remarks 231
1. Color and Spectroscopic Character of the Blood 232
2. Microscopical Examination of the Blood 237
1. Alterations in the Number and Appearance of the the Red Corpuscles . 238
2. Alterations in the Size and Form of the Red Corpuscles 240
3. Normal and Pathological Conditions of the White Blood-cells 242
4. Abnormal Additions to the Blood 245
Malaria Parasites 247
Chemical Examination of the Blood 250

CHAPTER VI.

Examination of the Digestive Apparatus.

Mouth, Palate, and Pharyngeal Cavity 252
The Mouth 252
The Lips 253
The Teeth and Gums 253
The Tongue 254
Mucous Membrane of the Mouth 255
Salivary Glands and Saliva 256
Microscopical Examination of the Contents of the Mouth 256
Microscopical and Bacteriological Examination of the Palate and Pharynx 260
Bacteriological Diagnosis of Genuine Diphtheria 260

PAGE

Examination of the Esophagus 263
Direct Palpation; Examination with the Sound 264
Examination of the Stomach 268
Anatomy of the Stomach 268
Inspection and Palpation of the Stomach 270
Percussion of the Stomach 273
The Halfmoon-shaped Space (Traube) 275
Auscultation of the Stomach 276
Illumination of the Stomach: Gastro-diaphanoscopy, ... 276
Examination of the Intestines 277
Inspection and Palpation 277
Percussion of the Intestine 280
Auscultation of the Intestine 281
Examination of the Peritoneum 281
Inspection of the Abdomen 281
Examination of the Liver 286
Anatomy 286
Inspection of the Liver 288
Palpation of the Liver 289
Percussion of the Liver 292
Pathological Relations of the Liver 294
Examination of the Spleen 296
Anatomy 296
Inspection of the Spleen 297
Palpation of the Spleen 297
Percussion of the Spleen 299
Pathological Relations of the Spleen 301
Auscultation of the Spleen 302
Examination of the Pancreas, Omentum, Retroperitoneal Glands 302
Examination of the Contents of the Stomach 303
Examination of the Process of Digestion 304
Preliminary Remarks upon Stomach-digestion and its Disturbances 304
Absorption 307
Method of Abstracting and Examining the Stomach-contents 312
Examination of the Stomach-contents 315
Results of the Examination of Stomach-contents, and their Significance . . . 323
Vomiting, and the Examination of What is Vomited 326
The Act of Vomiting 326
The Vomit 327
Examination of the Feces 332
Intestinal Discharges 333
Physical and Chemical Peculiarities of the Feces 335
Animal Parasites 340
Tape Worm 341
Round Worms 343
Trichina Spiralis 345
Microscopic Examination of the Feces 347
Chemical Examination of the Feces 353

CHAPTER VII.

Examination of the Urinary Apparatus.

PAGE

Examination of the Kidneys 355
Anatomy 355
Local Examination of the Kidneys 356
Pathological Conditions of the Kidneys 357
Differential Diagnosis of Tumor of the Kidney 359
Examination of the Ureters and Bladder 359
Examination of the Urine 360
(A) Normal Urine 362
(B) Pathological Urine 366
Anomalies in the Quantity 366
Color and Transparency of the Urine in Disease 368
The Specific Gravity of the Urine in Disease 372
Reaction of Urine in Disease 372
Pathological Odor of the Urine 373
Urinary Sediments 374
Sediments of Organic Bodies, or Direct Products of These 375
Inorganic Sediments 385
Examination of the Urinary Constituents in Solution 391
1. Anomalies in the Quantity of the Normal Constituents 391
2. Abnormal Constituents 393
Albumin 393
Bile-pigments and Bile-acids 400
Other Soluble Constituents of the Urine 406
The Urine as Affected by Medicines and Poisons 410
Examination of the Secretions of the Male Genito-urinary Apparatus 411

CHAPTER VIII.

Examination of the Nervous System.

Preliminary Remarks on Anatomy and Normal and Pathological Physiology 416
1. The Motor Tracts and Centers 416
2. The Sensitive or Centripetal Tracts 419
3. Centers and Tracts of the Special Senses 420
4. Remarks upon the Vessels Supplying the Brain 421
5. Topographical Diagnosis of Diseases of the Brain and Spinal Cord 423
Localization of Disease in the Spinal Cord 427
Method of Examination 430
Examination of the Seat of Disease 430
The Skull 430
The Spinal Column 434
The Peripheral Nerves and their Surroundings 435
Examination of the Condition of the Mind 436
Disturbances of Sensibility 439
1. Sensitiveness to Peripheral Irritation 439
(*a*) Cutaneous Sensibility 439
(*b*) Deep Sensibility 444
The Knowledge of Form (Stereognosis) 446

PAGE

2. Sensible Phenomena of Irritation and Pain from Pressure upon Nerves 446
1. Paræsthesia 446
2. Spontaneous Pain 446
Distribution of the Sensory Cutaneous Nerves 448
1. The Nerves of the Head 448
2. Nerves of the Neck and Trunk 449
3. Nerves of the Shoulder, Arm, and Hand 449
4. Nerves of the Lower Extremity 451
Disturbances of Motility 452
1. Paralysis 452
2. Disturbances of the Nutrition and Tone of Muscles 453
3. The Reflexes 457
1. Skin Reflexes 457
2. Tendon Reflexes (Periosteal, Fascial, Reflex) 458
4. Electrical Examination of the Nerves and Muscles 462
Regarding the Physics, and the Instruments Employed 462
Methods of Examination in General and their Physiological Results upon the Living Human Body 466
General Methods, and Explanation of the Terms Employed in Galvanic Examinations 467
Method of Examination in Detail. Normal Condition 469
1. Points of Stimulation 469
2. Method of Conducting the Examination 473
(*a*) Faradic Examination 473
(*b*) Galvanic Examination 475
3. What to Observe in Determining the Electrical Reaction 476
(*a*) Quantitative Excitability 476
(*b*) Qualitative Irritability of Muscles from Galvanic Stimulation . . 478
1. Reaction of Degeneration (RD) 478
2. Myotonic Reaction 483
3. Diagnostic Value of the Electrical Condition 484
(*a*) Significance of Reaction of Degeneration 484
(*b*) Significance of Diminished Irritability 484
(*c*) Significance of Increased Irritability 485
5. Mechanical Excitability of Muscles and Nerves 485
6. Co-ordination and Ataxia 486
7. Spasms of the Voluntary Muscles 488
8. Voluntary Muscles, their Innervation, their Function, and the Diseases that Disturb Them 492
1. Muscles of the Eye 492
2. Muscles of the Face 492
3. Muscles of Mastication, Tongue, Soft Palate 493
4. Laryngeal Muscles 494
5. Muscles of the Throat and Neck 495
6. Muscles of the Trunk 495
7. Muscles of the Thorax, Diaphragm, and Abdomen 496
8. Muscles of the Upper Extremity 496
9. Muscles of the Lower Extremity 500
Disturbances of Speech (Lalopathy) 502
I. Dysarthria and Anarthria 502
II. Aphasic Disturbances, Disturbance of Graphic Communication (of Mimicking and Singing) 503

PAGE

Mode of Procedure in Testing for Aphasic Disturbances 509
Sense Organs 513
The Eye 513
1. Movements of the Eye 514
2. The Pupils 517
3. Sharpness of Vision, the Color-sense, and the Field of Vision 519
Hearing 522
Taste 524
Disturbances of the Vegetative System in Nervous Diseases 525
1. General Phenomena 525
2. Disturbances of the Respiratory Apparatus 525
3. Disturbances in the Circulatory Apparatus 526
4. Disturbances of the Digestive Apparatus 526
5. Disturbances of the Urinary Apparatus 528
6. Disturbances of the Genital Apparatus 529
7. Disturbances of the Skin 529
8. Bones and Joints 530
Remarks upon The Diagnostic Value of the Symptoms in Nervous Diseases 531

APPENDIX.

1. Laryngoscopy 535
2. Rhinoscopy 543
3. Otosocopy 545
4. Ophthalmoscopy 546
5. Bacteria which come under Consideration in the Diagnosis of Internal Diseases . . . 548

INDEX 555

MEDICAL DIAGNOSIS.

PART I.

CHAPTER I.

INTRODUCTION.

The physician arrives at an opinion regarding his patient in two ways: by inquiry of the patient or of friends of the patient, and by his own objective examination. The result of the former is called the *Anamnesis;* the latter reveals the *Present Condition of the Patient.* The notes which the physician makes from time to time in the course of his continued observation of the patient, and in which he records the changing phenomena of the disease, constitute the *History of the Case.*

The judgment thus formed is briefly expressed as the *diagnosis.* In many cases this is pathologico-anatomical, since in a functional disease it assigns the case to one of the schemata which are used for this class of cases. Only in one part of the terms usually employed in specifying the diagnosis something is comprised which nowadays is of supreme interest—*etiology;* and at the present day medical science more and more seeks to establish an *etiological diagnosis.* Yet we know that many anatomico-pathological changes have very different causes, as, for example, diphtheritic disease of the mucous membrane, lobar pneumonia. This is also true of many functional diseases.

In making a clinical diagnosis, therefore, one must aim to have it comprise not only the anatomical or purely functional characterization of the disease, but that it should also include a statement of its etiology, if the present state of our knowledge in this direction enables us to do so.

But, furthermore, there belongs very much more to the conception of a diagnosis in its wider sense. Every person is individualized according to his physical development and his vital functions, but still more by the reaction of his tissues and his bodily functions to abnormal irritation. Hence every disease, according as it develops in this or that person, manifests a different, an individual, character. This fact is frequently observed, as in two cases of typhoid fever or two cases of pulmonary tuberculosis occurring in subjects apparently exactly alike,

in whom sometimes at the same stage of the disease there may be the greatest possible difference in the clinical picture. But very frequently, in order to ascertain how the disease manifests itself in an individual case, it is necessary to make a most careful general examination of the patient, to analyze the secretions and excretions, and often even to extend the observations over a considerable period of time.

The objective point of the physician's investigations at the bedside is therefore an *individual diagnosis*, first on purely scientific grounds, but still more important from the practical consideration that it must form the indispensable basis for individualizing the treatment.

In recent times we have learned a great many new facts regarding the etiology of diseases, and especially of those that are infectious. At the present time a number of the most important etiological diagnoses are made by microscopical and bacteriological means. Herein consists an extraordinary advance in clinical instruction. But in respect to what has been said above here lurks a certain danger. That is to say, in itself an etiological diagnosis is always schematic: for example, it is positively asserted that the exciting cause of a lung-disease is the tubercle bacillus, but this says nothing of the disease, tuberculous phthisis, which is present. In order to discover this the patient must be carefully examined from head to foot, and it must also be determined, partly by the anamnesis, partly by medical observation of the condition of the bodily functions and the variations in temperature. Formerly, before the tubercle bacillus was discovered, it was at least necessary to make a careful examination of the chest, perhaps also observations of the temperature, in order to make a diagnosis of tuberculosis. Thus, in a sense, one was compelled to give attention to the form of the existing disease. But nowadays it suffices for many, unfortunately, to find the bacilli in the sputum. This example serves to show how easy it is for the interest of the physician to be diverted from the patient himself by his investigations with the microscope and in the bacteriological laboratory. Every one who shares with me the opinion that it is necessary to make an *individual diagnosis* will take cognizance of the fact that clinical thinking may be neglected in the pursuit of these newer methods. The individual diagnosis can never be made at the study-table, but only and always at the bedside, and there only by a sort of artistic construction of the complete picture of the disease out of its collective phenomena, anatomical and functional.

Hence what was expressed in the Preface to the first edition of this work must here be repeated in the most emphatic terms: we should guard ourselves against the mistake of theorizing. A clinical diagnosis must always take into consideration the *whole man*. The clinician must never be satisfied with a diagnosis made with a microscope or a chemical reaction. All the phenomena must always be combined in a comprehensive description.

Since the chief object of this work is the teaching of the examination of patients and the presentation of the methods of conducting it, we limit ourselves to a short description of the method of obtaining the anamnesis.

ANAMNESIS.

What is it necessary for the physician to know, beyond what his examination reveals, in order to recognize a given disease in itself and to form a critical judgment regarding the patient in a larger sense? It is difficult to define this. Facts which appear insignificant in themselves, in experience often exercise a decided influence upon the special diagnosis, and especially in forming a judgment regarding the constitution of the patient or upon the timely recognition of a secondary disease. From having at hand clear knowledge of the symptoms of the different diseases, both of their remote or predisposing and of their directly exciting causes, a physician of experience is able in a short time to select what is essential from the past, and so to avoid too great prolixity. But it is always well for the beginner to secure as complete an anamnesis, or prior history, as possible, in order that he may allow nothing of importance to escape his attention.

The anamnesis generally begins with and involves the question as to whether the disease is acute or chronic, what organs are affected or are inclined to be diseased. This determines the examination to follow, in that certain organs are examined with greater exactitude than others. But the examiner must guard himself from too great influence or prejudice from the result of the anamnesis: the objectivity of the objective examination must be kept in view; and this, in turn, may give occasion for supplementing the anamnesis by propounding additional inquiries regarding certain occurrences and appearances, and thus a conclusion is finally reached. It is advisable for the student, under all circumstances, with all the patients he examines, and for the physician at least with his more important cases, to note down in regular order the results both of the anamnesis and of his examination. [See Translator's note, page 24 *et seq.*]

For the purpose of clinical instruction it is frequently of advantage to note the present state of the patient before making the anamnesis. We are thus better able to see the real facts and to preserve the objectivity of our judgment; but it cannot be expected that a physician will long continue to observe this rule in ordinary practice.

Mode of Taking the Anamnesis.—First, we always note the name, occupation, age, residence of the patient. Then we conduct, as simply as possible, a dialogue with the patient, or, in the case of a child or of a person who is insensible, unconscious, or mentally disturbed, with his neighbors or relatives. How much we may allow the person simply to tell, how much we must learn by asking questions, must depend upon the cultivation and intelligence of the one giving the information. We must particularly guard against asking the patient leading questions—that is, influencing the reply by the manner in which we put the questions. To the question: "Have you, then, really never had any pain in the bowels?" or, "Did you never have any pain in the bowels?" we shall almost certainly receive an affirmative answer, either from indifference, or from a desire to make his complaints as interesting as possible and to enlarge upon them, or, lastly, because he is of a very impressionable nature, and the question of pain suggests to him what in reality he has not had.

On the other hand, we must exercise close scrutiny of what the patient voluntarily communicates—a scrutiny which it is generally best not to allow the patient to know of. Where and how this has to be done can of course not be explained at length. We will here note only a few points which occur frequently:

(*a*) We must not accept without further inquiry the name the patient gives to a disease he has formerly passed through, since mischief is often done by the laity in the use of the names of diseases, as of diphtheria, typhus, etc. In any doubtful case we inquire its symptoms, and also what the physician who attended the patient had called the disease.

(*b*) The simulation of a disease is common. This was formerly confined in large part to the domain of hysteria; but, nowadays, from certain known social reasons, it is much more frequent. Neuralgia, rheumatism, trembling, spasms, even paralyses, the principal symptoms of traumatic neuroses, also pains in the bowels, asthmatic attacks, are the conditions which are most often simulated. In this way the physician may be led astray not only by false anamnestic statements, but clever persons are often capable of doing incredible things in simulating objective symptoms.

(*c*) The concealment of the existence of disease is manifest with reference to the different sexual diseases, especially syphilis. Women, moreover, often attempt to avoid all statements in regard to the sexual apparatus, even when it alone is diseased. Inebriates and those who practise onanism often confess their habits to the physician only with great reluctance.

What the Anamnesis Comprises.—The exact knowledge of the etiology and symptomatology of internal diseases is here the only correct guide, and at the same time gives us complete information respecting the cases which, under various circumstances, come under consideration. We are content with indicating the essential point of view by the introduction of a few examples. We may divide every anamnesis into the following two parts:

I. *Previous history of the patient:* This comprises all that it is important to know up to the beginning of the disease on account of which the patient consults the physician.

II. *The present disease:* This relates to the exciting causes, the commencement, and the course to the present time.

Here it is always necessary to ascertain exactly how deeply the present disease is rooted in the former history of the patient. That is to say, it is not only necessary to consider the first vestiges of the present disease itself, but also those morbid conditions which have prepared the soil for it.

Previous History of the Patient.

1. *Hereditary Disease* (*Heredity*).—This is of importance in so many diseases that in each and every case we have to inquire regarding the parents, brothers, and sisters of the patient, and also very often regarding the brothers, sisters, and parents of the parents. There especially come into view in this connection syphilis, tuberculosis, diseases of the

brain, and certain general neuroses. Heredity as regards rheumatism, carcinoma, diseases of the heart, and gout is of secondary importance, yet not immaterial. These diseases are in part inherited as such, in part they confer upon the descendants only the organic foundation, the disposition to the new development of the same or related diseases. Different descendants are variously divided by heredity. Often individuals, or a majority, are wholly exempt. It also happens that one generation is entirely passed over, and the trouble reappears in the following generation (hence the question regarding the grandparents).

It has been proved that most of the infectious diseases can be transmitted from mother to child *in utero*. This is true of syphilis, although the conditions are more complicated in this disease. Tuberculosis, as such, is only exceptionally transmitted.

2. *The manner of life, habits, profession, occupation, residence, experiences as to fatigue, other harmful influences to which they have been exposed, whether they have descendants, and, in the case of women, the number and character of their confinements*, compose this group.

Under *the manner of life* are considered the diet, character of dwelling, and the clothing. Injurious habits play a very important part in the manner of life, especially immoderate use of alcohol and other luxuries, of tobacco, narcotics, etc.; venereal excesses must also be taken into account. But it is important to remember that, within wide limits, the harm of these things differs with the individual.

Profession and *occupation* on the one hand affect the whole constitution, and on the other are often to be regarded as predisposing or exciting causes of disease; finally, they may exert a favorable or an unfavorable influence upon the course of an existing chronic disease. Thus, for instance, stonecutters and polishers, millers, workers in wool, by continually inhaling fine dust from the stone, flour, and wool, are very frequently inclined to bronchial attacks and diseases of the lungs; thus, too, the occupations that have to do with lead (type-setting, type-polishing, painting, etc.) or with mercury (making mirrors, etc.) frequently cause chronic poisoning by these metals. Persons who are engaged about sheep, swine, horses, or with the fresh skins and hair of these animals, are apt to have malignant pustule and other diseases. Phthisical patients are specially to avoid working as stonecutters, while victims of heart-disease are not to be employed with lead; and so of other diseases and occupations.

The *place of prior residence* is to be considered with reference to miasmatic (intermittent) endemic diseases or epidemics, which may have prevailed there at that time. With travellers, exotic diseases, which less frequently occur in their native places, as lepra, some infectious diseases, certain exotic animal parasites, etc., must be thought of.

As regards *fatigue*, army marches are to be regarded as particularly fruitful sources of disease. At the present time it is becoming more and more necessary to take into account those voluntary exertions which are connected with sport. A fall, slight perhaps, but whose effects continue; or a wound, without other immediate sequelæ except that it does not heal,—of these account must be taken, and also of very harmful momentary experiences, as sorrow, care, severe fright, anxiety.

Where there is *sterility* we consider anomalies of the sexual appa-

ratus of the man or woman, but especially the question of syphilis. The puerperal period, even when it does not pursue an unfavorable course, may in various ways be a source of disease.

3. *Diseases which one has had*—not only acute diseases, but the temporary outbreak of a chronic disease ending in apparent or real recovery.

Certain acute diseases may have as sequelæ certain other diseases which either are directly connected with them, as paralysis following diphtheria, nephritis after scarlet fever, or which appear after a shorter or longer period, as valvular disease of the heart from endocarditis in acute articular rheumatism, arising during scarlet fever.

The *outbreaks of a chronic disease* are often spoken of by patients as diseases which they have gone through; as, especially, the primary and secondary affections of syphilis, temporary manifestations of tuberculosis of the lungs, etc.

Some acute diseases are not prone to attack a person a second time. This is true of scarlet fever, measles, and typhoid fever. [But it is not uncommon for a person to have measles two, three, or even four times, and a second attack of typhoid fever is occasionally met with.] Certain other diseases, however, are liable to befall a person again, either because they leave behind a general disposition, the nature of which we do not understand, or because they produce some chronic local changes which give occasion for a new attack of the disease (erysipelas, malaria, pneumonia, articular rheumatism, appendicitis, and perityphlitis). Certain diseases of childhood are especially to be considered—for example, scrofulosis as early indication of tuberculosis; manifestations of hereditary syphilis; frequent convulsions as an early sign of anomalous condition of the nervous system. The diseases ordinarily designated as "children's diseases" generally have no significance as to the future, but yet sometimes, unfortunately, they leave lasting suffering behind them, as emphysema after whooping-cough, etc.

The Present Disease.

1. *The possible exciting causes* must be first considered. It is especially important for the early diagnosis of an infectious disease to inquire whether the patient has been exposed to infection. Many diseases are conveyed by a very short exposure; others require a longer exposure or even a personal contact. Also the period of incubation must be considered. This is the period from the moment of infection until the outbreak of the disease. With most transferable diseases this period is of a known, somewhat exactly defined, duration. Moreover, "taking cold," over-exertion, improper eating and drinking, taking of poison, etc. come under consideration.

It is to be remarked that the laity often assume something as an exciting cause, thus especially "taking cold."

2. *The first appearances and the course of the disease* up to the time of examination.

With chronic diseases the first appearances are sometimes, at the beginning, scarcely noticeable: they often consist only in a change from the previous behavior, unless the new condition in itself directly

appears to be one of disease: a person who previously had red cheeks becomes paler (all kinds of wasting diseases), a stout person without other reason becomes thin, one who always previously ate and drank little all at once eats and drinks considerably (diabetes), a person formerly very orderly becomes disorderly, forgetful (disease of the brain, especially progressive paralysis), etc. Even when they have made considerable progress, such gradually developing disturbances often are not at all noticed by ignorant and indifferent people.

CHAPTER II.

EXAMINATION OF PATIENTS.

The examination of the patient comprises—

1. *A general examination*, which takes into account certain phenomena of disease which concern the organism as a whole and are the expression of a pathological change of the whole organism.

2. *A special examination*, which inquires into the different regions and organs, the secretions and excretions of the body. At the bedside we generally proceed in such a way that, beginning at the head, we gradually go downward, in order to facilitate the investigation by examining contiguous organs. But in many cases it is better to group together organs that are functionally related, no matter what their anatomical location may be, since we thus quickly obtain a comprehensive view of the way in which the affected organs or systems are disturbed. Thus, in diseases of the heart, the heart and blood-vessels, in diseases of the nervous system, the central and different peripheral organs, are examined together. Sometimes, as in the case of very weak or very unruly patients, as children, the examination of the body must be very brief. Here the expertness of the physician especially is put to the test to the utmost degree.

It will best answer the purposes of study if the division of the subject throughout strictly conforms to the organ-systems, and hence the special part is divided into—

I. Examination of the respiratory apparatus.
II. Examination of the circulatory apparatus.
III. Examination of the digestive apparatus.
IV. Examination of the urinary apparatus, including also in part the sexual apparatus.
V. Examination of the nervous system.

Examination with the speculum and bacteriological diagnosis are discussed in the Appendix.

NOTE BY THE TRANSLATOR UPON KEEPING RECORDS OF CASES, AND A FORM FOR RECORDING THE RESULTS OF A MEDICAL EXAMINATION.

It is not practicable at the bedside to go through any set form for conducting the inquiry regarding the present illness. The most direct way of getting at it, and the one that will lead to the most satisfactory replies to our interrogatories, is to ask the question, What is your complaint? How are you sick? or some such direct question as this. In this way we get at once at the disease we are called upon first to diagnose and then to treat. As we proceed we will arrange the facts

in our minds, and when we make the record we shall place them in a natural and logical order. Having a regular form for keeping records of cases soon develops an order of procedure in accordance with it.

Case-taking is a most valuable aid to the student in clinical study.

1. He learns to make a systematic examination of the patients he sees. He forms the habit of bringing before his mind each factor in the case in orderly succession. There are two advantages from this: First. He forms the *habit of thoroughness in examining* his cases. Second. He can readily compare one case with another, having arranged the factors of each in like order. While it is not necessary in making the examination to have or to follow strictly a printed form, yet it is desirable to have some regular form for making the record, so that cases that are similar can be readily compared. One case may require going over only a few points; in another it will be necessary to examine every organ in the body.

2. The memory is greatly strengthened. Memory depends upon attention and repetition. Case-taking cultivates both of these in an eminent degree. Facts and symptoms that else would escape notice entirely or be only slightly noted are brought prominently before the mind for consideration. Their value or bearing is weighed, and so they are strongly impressed upon the mind.

3. The mind is developed by this habit of carefully reflecting upon every feature of a case. Thought is both stimulated and made easy. Clearness and power of thought are increased. Independence of judgment is cultivated. Both knowledge and intellectual cultivation are acquired. "By knowledge is understood the mere possession of truths; *by intellectual cultivation or intellectual development, the power acquired by exercise of the higher faculties, of a more varied, vigorous, and protracted activity*" (Sir William Hamilton).

4. Ease and habit of writing are almost unconsciously acquired. This is most valuable. The great majority of physicians keep no records of cases. Many never record or publish important ones, because they have not the facility of writing which comes with practice. *Anything is easy to the practised hand.* "Who can estimate how much we have lost from the fact that generations of men gifted with powers of acute and shrewd observation have passed away without leaving one record behind them? Think not that it is the hospital physician or surgeon alone who can advance the progress of medicine. There is not a practitioner who could not aid this great work. But he can only add to it with efficiency if he has *faithfully recorded his observations*, and does not trust to the general and vague impressions of unassisted memory. Therefore, on all grounds, personal to yourselves and general for medical science, so engrain this habit within you that it becomes a second nature" (Coupland).

THE ANAMNESIS.

Personal and Previous History.

Name, Address,
Birthplace, Age, Sex,
Family history—Heredity:
Father,
Mother,
Brothers,
Sisters,
Other relatives.
Manner of life, habits, occupation, residence, etc.
Previous diseases—character and results.
(Note each one that was of such a character as to have any lasting effect upon the health or vitality.)

Present Illness.

Duration,
Possible exciting cause,
How began—suddenly or not; prodromal symptoms,
Course of the disease till the time of examination.

Examination of the Patient.

General examination:
Appearance,
Psychical condition,
Position in bed,
Structure and nutrition,
Skin and subcutaneous tissues,
Temperature,
Pulse.

This covers the general features of every case. Attention has been directed, by what has been learned thus far, to some one or more of the special organs or systems of the body, It is usually best first to examine that, and to make this examination very full and thorough. Then the remaining organs of the body can be examined with greater or less fulness according as they are found to be affected by the principal disease or as they are related to the one specially diseased. It is well to form the habit of following a certain order in examining each organ. One is much less apt to overlook any part; and, too, as has already been pointed out, the records will be more easily consulted and compared. For this purpose it is well to take the order of the text-book, so as to become thoroughly familiar with each subject. It is not of so much importance that this or that one be adopted, provided it is a good one. But we have here a notable illustration of the truth and value of the Spanish proverb: "Beware of the man of one book."

Presuming that those who use this work will follow the order laid down in it, the form now given conforms to the order in which the systems are treated.

SPECIAL EXAMINATION.

Examination of the respiratory apparatus:
- Nose,
- Larynx.

Examination of the lungs:
- Inspection of thorax,
- Palpation of thorax,
- Percussion of thorax,
- Auscultation of lungs,
- Auscultation of voice,
- Measurement of thorax,
- Cough and expectoration.

Examination of circulatory apparatus:
- Inspection and palpation of the region of the heart,
- Percussion of the heart, Apex-beat,
- Auscultation of heart,
- Examination of the arteries and veins,
- Examination of the blood.

Examination of the digestive apparatus:
- Mouth, gums, and pharynx,
- Esophagus,
- Stomach,
- Intestines,
- Peritoneum,
- Liver,
- Spleen,
- Pancreas, omentum, retroperitoneal glands,
- Contents of the stomach and vomited matters,
- Process of digestion,
- Feces.

Examination of the urinary apparatus:
- Kidneys,
- Ureters and bladder.

Examination of the urine:
- Amount in twenty-four hours,
- Reaction, Odor,
- Specific gravity,
- Sediment,
- Albumin,
- Blood,
- Bile,
- Sugar,
- Other constituents.

Examination of secretions of the male sexual apparatus.

Examination of the nervous system:
- Disturbances of sensibility,
- Motor disturbances,
- Disturbances of speech,
- Organs of special sense.

PART II.

CHAPTER III.

GENERAL EXAMINATION.

This consists of a number of subordinate divisions—namely, we have to consider—

I. The psychical condition of the patient.
II. The position in bed, attitude, posture.
III. The general structure of the body and the nutrition.
IV. The skin and the subcutaneous cellular tissue.
V. The temperature and the pulse.

I. THE PSYCHICAL CONDITION OF THE PATIENT.

From this—that is, from the clearness of his intelligence, his susceptibility to external impressions, his power of thought, from the possible presence of depression or irritability—we may often obtain important points of diagnosis, both for diagnosis in the narrower sense, certain diseases being accompanied with definite manifestations of this kind, and for diagnosis in a broader sense—that is, to form an exact opinion of the present disease—since the severity of a disease, the possible turn for better or worse, often becomes manifest by the psychical condition of the patient.[1]

II. THE POSITION OF THE PATIENT, ATTITUDE, POSTURE.

The position and attitude of the patient furnish a very simple aid to diagnosis, because generally they can be determined by a single glance of the eye. From them conclusions in various directions may be drawn. People in health or only slightly sick usually assume the dorsal position or a position upon one side in a certain unconstrained, comfortable position (the active dorsal or side position). On the contrary, patients who either are not wholly conscious, or who have become very weak, frequently are inclined to slide down toward the foot of the bed and sink into a heap there—a position which manifests weakness, and in some respects, but especially for breathing, is very unfavorable (the *passive dorsal and side position*).

In acute infectious diseases, more than elsewhere, the *passive dorsal position* is specially noteworthy. It is particularly so when apathy and clouded intelligence are combined with great muscular weakness,

[1] Regarding this and the way in which the examination in this direction is conducted, see the section on Examination of the Nervous System.

as is frequently the case in typhoid fever, where such a condition of the patient is so frequently and sometimes early present that it may aid in the diagnosis.

But in still another way *the position in bed* is sometimes characteristic. Patients with *acute affections of the chest-organs* involving only *one side* (pneumonia, pleurisy, pneumothorax) generally *lie upon the side*, and for the most part *upon the side affected*. This may be due to various causes. The pain caused by breathing is generally in this way diminished, because by lying upon the side the motion of that side is very much lessened, while, on the other hand, the motion of the upper side in breathing is greater when on the side than when the patient lies upon the back; hence the sound side, when the patient lies upon the diseased side, can better compensate for the loss of the portion diseased. In exudative pleuritis frequently there is the further advantage in lying upon the affected side that the exudation least interferes by pressure with the healthy side.

Yet patients with pneumonia not infrequently lie upon the healthy side because they are not able to endure the pressure of the weight of the body upon the diseased side. That in diseases of the chest patients are generally inclined at the beginning of the disease to lie upon the sound side, and later upon the diseased side, I am not able to affirm.

Children sick with typhoid fever sometimes lie on one side with the legs drawn high up (position of a hunting dog). Such patients, being often at the same time in stupor or unconscious, continually return to this position. It seems like an unconscious impulse, and at the same time has something of the so-called compulsory position.

Difficult breathing (*dyspnea*), if extreme, prompts one to assume the upright sitting posture in bed or in an easy-chair—*orthopnea*—because in this attitude the action of the accessory muscles of respiration is more effective than when lying down. Orthopnea may therefore occur with all diseases which are accompanied with marked interference with respiration, as in narrowing of the air-passages in disease of the lungs (comparatively rare with phthisis),[1] in diseases of the pleura, heart, pericardium, with large effusions into the abdominal cavity which press the diaphragm up, and in general dropsy with effusions into the cavities of the body. In the severest cases the patients may indeed be obliged to keep the sitting posture, even to sleep. The continued exertion of sitting and the diminished sleep obtained in this position, besides the great anxiety and excitement these patients generally have, usually quickly bring on exhaustion.

Another group of characteristic situations and positions in bed refer to *diseases of the brain and its membranes*. Thus meningitis betrays itself often at the first glance by *opisthotonos*, with the head boring into the pillow—so-called contraction of the neck. In circumscribed disease of the cerebrum the head is sometimes persistently inclined to be drawn forcibly to one side—forcible contraction of the head. In affections of the cerebellum and of the pediculi cerebelli medii the patient sometimes continually keeps a certain lateral position as by an unconscious or only half-conscious impulse. If the patient is moved

[1] See under Dyspnea.

out of this position, he always returns to it again immediately. Such positions are called *compulsory positions*. It is well to use this expression exclusively for those attitudes and positions which the patient takes or remains in instinctively when there is complete exclusion of consciousness. But the positions of patients suffering from disease of the chest, etc., mentioned above, do not strictly belong to the compulsory positions.

Paralyses and atrophies of the most different muscles, particularly those of the trunk, of course furnish a great number of anomalies.[1] There is a sign which belongs to the most varied chronic inflammatory abdominal affections which involve the peritoneum: sometimes patients have a peculiar bent-forward attitude in standing and walking, because in an upright attitude they feel tension in the abdomen. Here belong parametritis, more severe chronic perityphlitis, etc.

III. THE STRUCTURE OF THE BODY AND NUTRITION: WEIGHT.

The development of the skeleton determines the form of the body. Generally, firm bones and broad, flat chest are characteristics of strong and enduring health, while those persons of delicate skeleton, especially with slender ribs and narrow chests, are considered capable of both limited life and endurance. Yet this is only a general rule. We often see people of delicate build who are remarkably tough and enduring, both with reference to exertion and disease; and not infrequently we find robust people with little power of resistance, especially to acute diseases.

Unusually defective development of the skeleton, also other anomalies of the growth of the skeleton similar to rhachitis (among others "the fetal rhachitis"), are frequently found in idiots and cretins. There is also the development known as *dwarf*, without any other anomaly.

The form of the thorax is of especial importance. With a slight and narrow chest-cavity there is a proportionally frequent disposition to tuberculosis of the lungs; and, on the other hand, a certain fulness carries with it a tendency to emphysema of the lungs.[2]

The significance of the structure of the pelvis is manifest in the practice of obstetrics.

The muscles, the subcutaneous tissues, and the skin furnish a means of judging of the nutrition and also of the weight. In general, well-nourished and healthy persons have a certain volume and firmness of muscles. There is also a relation between the muscles and the skeleton. But even in perfectly normal persons there is a very marked difference in the volume of the muscles, which is not always explained by differences of occupation. By experience the eye gradually becomes quick in recognizing a suspiciously small muscular volume; yet the firmness of the muscles is a better guide to an opinion than their volume.

The fat of the subcutaneous tissues may be very differently developed in persons of good health. As a rule, it varies with the age,

[1] See section on Nervous System.

[2] This will be more particularly spoken of under Respiratory Organs.

being greater for the first years of life up to the forty-fifth or fiftieth year. Beyond this it again, as a rule, becomes less. It also sometimes varies in a shorter time without being caused by disease, most frequently and markedly in women at about twenty years of age. It varies also, as a matter of course, with the kind and the richness of food, as well as with the occupation. Loose adipose tissue generally indicates a weak organization.

A marked degree of leanness of the subcutaneous tissue under all circumstances is suspicious, and suggests an examination as to whether it may be caused by disease. In the same way the accumulation of fat beyond a certain degree becomes pathological. The measure or degree can only be established by experience.

Of much greater importance is a *commencing*, even though a slight, wasting away of the subcutaneous fat, and eventually also of the muscles. As we have said, this is sometimes physiological. It can also take place from very poor nourishment, as among the poorer classes. But in the majority of cases it is caused by disease, and it is therefore important not to overlook it. This wasting can only really be learned by the physician when he has known the patient for some time. When this is not the case he must rely upon the statements of the patient and his surroundings, and therefore this subject properly belongs to the "previous history." When the emaciation is marked, its proof is furnished by the condition of the skin. In these cases the skin of the patient's whole body is loose, and can easily be taken up in folds.

Excessive wasting is denominated *atrophy*, *emaciation*, and, when this is accompanied by general loss of strength and failure of function, *marasmus* or *cachexia*.

Weight.—The weight of the body is an excellent index, and one which is superior to all other signs of corpulence and its increase or diminution. The absolute value of the weight of the body in the different periods of life has no diagnostic interest, for the reason that it varies within wide limits. Likewise the relation of the weight of the body to the height and the circumference of the chest has scarcely any significance for our purposes, because as yet a norm has not been determined. On the other hand, change in the body-weight wrought by disease is of the greatest importance. In chronic diseases this is an extremely valuable means of determining whether the disease is increasing, standing still, or is being recovered from. Taking the weight regularly (say, weekly) in cases of tuberculosis is especially to be recommended, also in diseases of the digestive apparatus. In convalescence from acute diseases, following the weight of the body is also a very important aid, especially for the early recognition of the possibility of the disease becoming chronic or of the presence of associated chronic diseases.

Edema influences the weight in a peculiar manner. It causes a misleading increase in weight when it makes its appearance, and a by no means disagreeable decrease of the weight of the patient when it disappears. It is important in diseases which dispose to dropsy to always think of its possibility whenever striking alterations of weight appear, even in cases where edema or effusion cannot be demonstrated,

because the lesser degrees of anasarca generally elude exact clinical demonstration.

According to Bornhardt,[1] the relation of the weight of the body, P, to the height, H (in cm.), and to the average circumference of the chest, C (measured at the level of the nipples, in cm.), for the average individual, may be reckoned as follows:

$$P = \left(\frac{HC}{240}\right) \text{kilograms.}$$

The weight of the body of the newly-born and its increase during the first months is of special significance.[2]

Diseases of the alimentary tract and all febrile diseases, whether acute or chronic (of the latter especially tuberculosis), also severe forms of diabetes mellitus, and, finally, all malignant growths, produce marked emaciation. But a certain degree of emaciation can be produced by any disease of an internal organ.

IV. SKIN AND SUBCUTANEOUS CELLULAR TISSUE.

In medical diagnosis the condition of the skin and subcutaneous tissue is considered with reference to the following points:

A. The condition of general nutrition.
B. The moisture of the skin; perspiration.
C. The color of the skin.
D. Certain pathological appearances of general diagnostic value (characteristic eruptions, hemorrhages, scars, etc.).
E. Edema.
F. Emphysema of the skin.

Skin-diseases proper and certain acute infectious diseases with special localization upon the skin (the so-called acute exanthematous diseases) are not considered in this work or only incidentally mentioned.

A. The State of Nutrition of the Skin.

In extremely old age the skin over the whole body appears to be physiologically thinner, the subcuticular cellular tissues probably having the greatest share in this atrophy. In earlier years a noticeable general atrophy of the skin or the subcuticular cellular tissue exists only where there is a very severe cachexia. The skin is then thin and generally dry. It loses its tone, and when taken up in a fold resumes its place slowly.

The different forms of circumscribed atrophy of the skin which have been described do not interest us here. They belong to works upon skin-diseases.

B. The Moisture of the Skin; Perspiration.

Physiology teaches us that the moisture of the skin, as well as the visible secretion of perspiration, is influenced by various circumstances.

[1] Cited by H. Vierordt.

[2] Regarding this subject see works upon obstetrics and diseases of children, also *Daten und Tabellen*, by H. Vierordt.

It is increased during active exertion by increased temperature of the blood, by moist heat, by mental impressions, especially fear; finally, by certain ingesta, as hot tea, by pilocarpin, etc. In some of these cases there is at the same time an increase of heat of the body, which is overcome by the perspiration, cooling being caused by its evaporation. It is well known that the perspiration exercises a continual regulating influence on the temperature of the body.

The loss of water by evaporation (the greater part of the insensible perspiration) in health is, *cæteris paribus*, greater at night than during the day. It seems to alternate with the secretion of the urine.

In healthy people the secretion of perspiration is in this way very changeable. But it is still more so in cases of illness. It may be increased to such a degree that the whole bed may be wet through (*hyperidrosis*). On the other hand, it may be so diminished (*hyphidrosis*) that the skin is perfectly dry (*anidrosis*). Hyperidrosis of the whole body is called *hyperidrosis universalis;* if confined to a part of the body, *hyperidrosis localis*. The latter may be unilateral (*hemidrosis*).

The influences which produce these morbid alterations of perspiration are without doubt of different nature. In the first place, there are chemical influences by abnormal products in the blood and lymph, as, for instance, accumulation of carbonic acid; of urinary products, as urea; products of muscular exertion. Here also belong the auto-infections. Then there are bacterio-chemical bodies, and, lastly, other poisons, which produce or suppress perspiration.

These factors in part act directly upon the sudorific glands, in part indirectly through the nervous system. The latter, however, may be acted upon by independent influences, giving rise to the following: psychical perspiration; hyperidrosis and anidrosis in central and peripheral diseases of the nervous system.

A general perspiration may take place in cases of illness—

1. When there are present conditions which are analogous to those which produce it in persons in a state of health, as in cases of strong tetanic convulsions by the increased muscular work and heart-action. On the contrary, in cases of epileptic, hysterical, and other convulsions we have either no perspiration, or at least none corresponding with the very great muscular exertion; in all possible diseased conditions connected with great excitement, especially fear, or with severe pain; and again, sometimes, not always,[1] from a high degree of atmospheric heat, warm baths, moist warm pack, or sudorifics (pilocarpin, etc.). Morphin also, with some persons, induces perspiration.

2. *In difficult breathing—dyspnea.* This is generally accompanied by sweating. In the same way sweating sometimes occurs with heart-disease, accompanied by an engorged condition of the "greater" circulation; also with all diseases of the respiratory organs and their surroundings which interfere with respiration. Perspiration is here produced both by the venous quality of the blood and the anxiety or fright[2] which is always present in dyspnea.

3. *In febrile diseases.* Sweating usually occurs with the fall of the temperature in these diseases. In these cases the perspiration performs

[1] See below under Anidrosis.

[2] See above.

the additional service of washing away the poisons which have accumulated during the course of the disease. The most important instances are (*a*) the critical sweat of a rapid definite decline of the fever, especially frequent in pneumonia and febris recurrens [relapsing fever]; (*b*) the sweat which regularly accompanies the fall of temperature in intermittent fever and pyemia (diseases which manifest themselves by rapid rise and fall of temperature), the night-sweats of the hectic fever of phthisis, and the sweat of the remittent (hectic) fever of typhoid fever; and (*c*) the cold sweat of collapse (that is, the sudden failure of strength in the death-struggle).

Acute articular rheumatism manifests itself by considerable poisonous perspiration, which may not depend upon a fall of temperature; this is also true in rachitis. Finally, there is always the inclination to perspiration in the commencement of convalescence from severe diseases and in parturient patients, when there is great weakness and the vascular system is easily excited.

Local sweating occurs in various neuroses, also in organic diseases of the nervous system. There is very frequently sweating of the whole of one side (*hemidrosis*) or of the head alone, as in Baselow's disease, migraine, hysteria, localized disease of the brain, and in mental diseases.

Diminished secretion of sweat, even to complete *anidrosis*, is observed chiefly in high continued fever. It is, moreover, a peculiarity of all diseases which are accompanied with considerable loss of water by the bowels or the kidneys, of severe diarrhea of any kind, contracted kidney, and diabetes. The anidrosis which exists with general dropsy, in consequence of the anemia of the skin produced by the pressure and stretching, has a peculiar appearance.[1]

The anidrosis of high fever and general dropsy is very persistent, sometimes resisting all therapeutic measures, as, for instance, those acting directly upon the skin (moist heat, etc.), and the medicines already mentioned.

Qualitative alterations of sweat exist sometimes in severe jaundice,[2] when it contains the coloring matter of bile and is yellow in color; also, when the urinary secretion is greatly diminished or entirely suppressed, as in nephritis, diseases of the urinary tract, and cholera. It then sometimes contains considerable quantities of urinary products, which in some cases, by the evaporation of the perspiration, crystallize upon the skin (especially upon the nose and forehead) in small white scales. This is called *uridrosis*, the scales giving the reaction of urinary ingredients. Occasionally, however, they consist only of common salt.

C. The Color of the Skin.

As is well known, races differ in the color of the skin, but even in the Indo-Germanic race there are variations depending upon the stock, the climate (blond, brunette). In some nations the pale, in others a more florid, complexion, especially of the face, preponderates. We know that there are differences depending on the mode of life; also that, even as regards the so-called healthy color of skin, considerable

[1] See under E.

[2] See under Icterus.

individual variations exist. But, after all, the hue of the skin stands in intimate relation to a large number of diseases of the internal organs.

It is considered most suitable to judge from the color of the countenance, the portion of the skin most generally reddened; and, since on every hand we have opportunity for practice, it is well to sharpen the eye for critically examining this part of the body. But the color of the countenance can sometimes deceive us,[1] and it is therefore advisable always to examine the mucous membrane of the lips, mouth, and throat,[2] and, besides, to observe the color of the skin of a part of the body usually covered by the clothing.

We recognize the following abnormal colorations of the skin:

1. The pale skin.
2. The abnormally red skin.
3. The blue-red cyanotic skin.
4. The yellow skin of icterus.
5. The bronze skin.
6. The gray skin produced by nitrate of silver.

1. The Pale Skin.—This can to a certain extent be physiological, especially in persons who spend little time in the open air. In these cases a glance at the mucous membrane gives further information. But one can be deceived regarding such persons, who, having exposed the face (also arms and hands) frequently to radiant heat or to cold and heat in rapid succession, often have a local redness of face. This redness of face may arise from other causes.[3]

Only experience can enable one to distinguish between physiological paleness and that produced by disease. The recognition of the latter is frequently aided in that it is associated with a grayish, yellowish, or, in a word, with a sickly, color. The color of the skin is produced by the fulness of its capillary vessels. The abnormal paleness may be dependent upon disturbance of the circulation, and in consequence of diminished force of the heart or active narrowing of the peripheral arteries, or by a lessening of the quantity of the blood-constituents, chiefly of the hemoglobin.

The redness of the skin depends upon the degree to which its capillaries are filled with blood. There is abnormal paleness if either too little blood or too light blood is circulating in the capillaries of the skin. The causes of the morbid paleness are, therefore, on the one hand, disturbances of circulation—*i. e.* decrease of motor power of the heart as well as arterial spasm, or, on the other hand, deficiency of hemoglobin. It is an important diagnostic point to decide in all cases of paleness, first of all, to which of these two principal groups the paleness belongs. The surest diagnostic means, however, and the one which should always be applied if there is the least doubt, is the examination of the blood.[4]

We distinguish (*a*) *Temporary paleness*, which is partly physiological and partly pathological. It occurs with strong emotion, especially fright;

[1] *Vide* especially under Red Skin.

[2] It is wrong in making a diagnosis of anemia to include the observation of the conjunctival mucous membrane. It is not decisive, since many persons in whom the teguments are elsewhere pale, at times easily have the conjunctiva injected.

[3] See under Red Skin.

[4] See under Examination of the Blood.

in syncope or fainting; in the chill of fever, which ordinarily accompanies a rapid, considerable elevation of temperature; in spasm of the capillary vessels; in vascular spasm which occurs spasmodically, particularly in the extremities. This spasm is observed either as a simple vaso-motor neurosis or in connection with certain phenomena in the heart.[1] (*b*) *Paleness lasting a longer or shorter time.* This comes on sometimes quite rapidly, at least in the course of a few moments, during profuse hemorrhage and in sudden collapse—that is to say, in sudden failure of the heart as it occurs in acute, and sometimes chronic, diseases, and in acute poisoning. The sudden paleness in consequence of the loss of blood or collapse is accompanied by acceleration and attenuation of the pulse, great weakness, and sometimes with disturbance of consciousness.

External hemorrhages make themselves evident. But cases of severe internal hemorrhage, especially of the stomach or bowels, of ruptured aneurysm, hemorrhage from internal wounds of any kind, are declared only by this sudden paleness, sometimes even before the patients themselves, if quiet in bed, complain of weakness.

In a case of endocarditis which I saw the patient became pale, as one does from an internal hemorrhage, with increased frequency of pulse and stupor, within less than ten minutes. At the autopsy there was found a recent total rupture of an aortic valve.

This paleness, spoken of under (*b*) above, can develop more slowly within a few hours or days by considerable repeated hemorrhages. In such a case the examination of the blood always shows it to be watery, deficient in hemoglobin, and often also in red corpuscles, because after losses of blood the watery constituent is always restored first. [This condition is called *hydremia.*] It develops as a symptom of weakening of the heart's activity in all acute and chronic diseases of the heart and pericardium; also in diseases of parts adjacent to the heart, as pleurisy and abdominal affections, with much pressure upon the diaphragm in case they interfere with the action of the heart; finally, in many acute diseases, especially in diphtheria, in heart-failure from diseases affecting the muscular structure of the heart, and very often and very quickly in acute catarrh of the stomach (acute dyspepsia). Here hydremia is connected with imperfect fulness of the blood-vessels.

Finally, paleness of the skin comes on in certain conditions generally unnoticeable, insidious, and is a chronic condition; in the so-called *special diseases of the blood and of the blood-making organs*—indeed, most unfortunately, from a diminution of the hemoglobin, hence in chlorosis, also in pernicious anemia, leukemia, pseudo-leukemia. In this list also probably belongs malarial cachexia. Paleness is a symptom of all slowly-developing *secondary anemias* (*cachexia*) as they occur in a large number of diseases, such as all chronic febrile diseases, especially tuberculosis; in suppurations without fever; in continuing slight hemorrhages, as in many tumors and in ankylostomiasis [Egyptian chlorosis]; in all chronic diseases of the digestive tract; in most diseases of the female generative organs; in the different forms of chronic nephritis, especially the large white kidney; in

[1] Compare section on Circulatory Apparatus.

chronic poisoning, especially by mercury or lead; sometimes, also, in constitutional syphilis; in malignant growths, especially in cancer proper; and in *chronic diseases of the heart*, but especially in fatty heart and mitral and aortic stenosis.

In the first two of these three groups the coloring faculty—*i. e.* the faculty of the blood to redden the skin—is always more or less diminished; but also the defective power of the heart may contribute to paleness. In the third group, that of heart-diseases, however, the principal causes are defects of the circulation.

Often there exists not only paleness of the skin, but its color has a still further characteristic appearance. In severe anemias we have a peculiar waxy appearance, which not rarely has a yellow tone. A striking, light white skin often exists with the so-called large white kidney (chronic parenchymatous nephritis), also in a certain proportion of the cases of lead-poisoning (which latter is often of a grayish white), of leukemia, and of tuberculosis. In chlorosis the skin has a greenish hue; in diseases of the heart-muscle and in mitral insufficiency the skin is generally a smutty yellow, while in the cachexia of cancer it is often gray-yellow.

Often a large development of adipose tissue strikingly contrasts with a pathological paleness, and this is especially to be seen in diseases of the blood, particularly in chlorosis and pernicious anemia and also in heart-diseases. In both cases, however, one must be careful not to be deceived by the presence of edema.[1]

2. Abnormal Redness of the Skin.—This expression comprehends a superfluity of normal blood, because up to the present time we do know of such a condition—*i. e.* a genuine plethora.

General abnormal redness of the skin is always a sign of a general hyperemia of the cutaneous capillaries, and it occurs in high fevers, especially in continuous fevers. It also is present during the perspiration following a warm bath. Finally, in poisoning with atropin, even in very mild cases, it is developed like the redness of scarlet fever. (The scarlet-fever redness, being connected with a disease of the skin, does not belong here.)

Local redness, depending upon a dilatation of the capillaries, exists very frequently in the face, and indeed is physiological in those who labor in the sun. It comes and goes quickly, as in blushing (*rubor pudicitiæ*), in nervously excitable persons in consequence of very slight psychical impressions, also not infrequently as a result of physical exertion. Moreover, we see redness of the face in fever; finally, one-sided redness of face in the "paralytic" form of hemicrania.

Tuberculosis is characterized by a very marked variation in the fulness of the capillaries of the face: if the patients are entirely at rest and without fever, they are generally pale, but under excitement or exertion, after eating, and, lastly, during fever, they exhibit a very striking, generally bright, redness of the cheeks and often a sharply-defined spot (hectic redness).

In the slight forms of anemia, especially if associated with nervous irritability of heart, likewise with local vaso-motor disturb-

[1] Compare p. 47.

ances, there is sometimes intense redness of the face which may conceal the anemia from the physician.

For distinction of circumscribed hyperemia from Hemorrhage in the Skin, see under the latter.

3. The Blue-red Skin, Cyanosis.—This is most plain on the parts that normally are bright red, hence more than elsewhere on the mucous membranes, on the lips, cheeks, etc.; also on the knees, the phalanges of the fingers, and under the finger-nails. A moderate degree of cyanosis, therefore, would only be discovered at these parts. A marked degree, on the other hand, exhibits a blue color spread over the whole body, while those parts, especially the mucous membrane, become black-blue.

The cyanosis of the new-born, with heart-failure, is so striking to the experienced observer that it is regarded by him as pathognomonic. This symptom occurs, according to the gravity of the organic changes, either persistently or only after exertion. The popular name for it is "blue disease." One only sees anything like it in the death-agony and, exceptionally, in severe spasms with marked interference with breathing. The combination of cyanosis with great paleness is designated as "livid skin."

Cyanosis arises from the blue-red color of the capillaries, and this, as is well known, is caused by an accumulation of carbonic acid and deficiency of oxygen—that is to say, by the venous or hypervenous character of the capillary contents.

Carbonic acid in the blood (serum and red corpuscles) arises from —1. Interference with the exchange of gases in the lungs; 2. From the slowing of the capillary circulation and the consequently diminished gas-exchange in the tissues—that is to say, the diminished giving up of CO_2 by the tissues to the blood.

Cyanosis arises, therefore—1. In disturbed respiration and circulation through the lungs; 2. In disturbance of the "greater circulation," which may be general or circumscribed according as the stoppage may be general or local. The two causes may be combined.

Here belong to 1—

(*a*) *All conditions which cause a narrowing of the larger air-passages or of a large number of small bronchi:* inflammation of the neighborhood of the pharynx or entrance to the larynx; retropharyngeal abscess, angina Ludovici; very exceptionally a diphtheria of the throat. (In all of these cases the interference with respiration is either direct or dependent on edema of the glottis.) Here belong also those rare, sudden obstructions of the pharynx by foreign bodies, as a piece of meat and the like. The following are enumerated: spasm of the glottis, paralysis of the dilator of the glottis (crico-arytenoideus post.), all acute and chronic inflammations of the larynx, but especially croup; tumors of the larynx; cicatricial narrowing of the larynx; foreign bodies in the larynx (something swallowed or vomited) and wounds of this organ; also foreign bodies, croup, and scars in the trachea or one or both primary bronchi; compression of these from without by enlarged glands; aneurysm of the aorta; mediastinal tumors, etc.; severe diffuse bronchitis, especially the acute croupous form; bronchial asthma.

(*b*) *All diseases of the lungs and diseases of the chest-cavity and its neighborhood which hinder the expansion of the lungs or wholly compress them:* emphysema of the lungs; all forms of consolidation; pleuritic and great pericardial exudation; pneumothorax; tumors in the chest-cavity; abdominal diseases with marked upward pressure of the diaphragm.

In these conditions there exists also a disturbance of the pulmonary circulation, apart from the immediate interference with respiration. In emphysema a great many capillary blood-vessels are obliterated, which is also the case in tuberculosis and chronic pneumonia. Severe exudative pleurisy and pneumothorax, on the other hand, produce obliteration of capillaries by compression. The afflux of blood to the respiratory surface of the lungs is in such cases consequently always diminished, and this is an additional cause of dyspnea.

(*c*) *Paralysis of the respiratory muscles:* bulbar paralysis; peripheral neuritis; paralysis of diaphragm from peritonitis; spasm of the muscles of respiration; epilepsy, tetanus, but, on the other hand, very rarely hystero-epilepsy; special muscular diseases; myopathic forms of progressive muscular atrophy, trichinosis, myositis ossificans.

Disturbances of the circulation through the lungs occur in a number of the diseases which interfere with respiration. In emphysema a large number of capillary channels are closed, also in tuberculosis and other chronic lung affections; a large pleural exudation not only compresses the lungs, but also the capillaries. This acts in the same way as a hindrance to respiration.

(*d*) *Diseases of the heart which result in obstruction of the pulmonary circulation.* In several of these conditions, especially inflammatory diseases of the pleura, of the peritoneum, in trichinosis of the diaphragm, the insufficient breathing, as well as the cyanosis, will be increased by the pain caused by the act of breathing. This circumstance is of great practical utility, because a part of the dyspnea, as well as of the cyanosis, can be removed by alleviating the pain which is the result of the act of respiration.

In persons very much wasted, especially from tuberculosis, cyanosis may be absent even in spite of the loss of a large part of the breathing-surface of the lungs, since the remaining normal portion suffices for supplying the required quantity of oxygen to the diminished quantity of blood.

Under heading 2:

Slowing of the blood-current in the capillaries of the greater circulation is dependent upon stopping of the venous outlet. This can be general, and caused by all the conditions of the first category, general cyanosis, or it can be occasioned by a venous stopping of an extremity or of the head, and so produce a local cyanosis.

General venous stasis occurs in diminished motive power of the right ventricle (valvular deficiency, congenital stenosis of the pulmonary artery, diseases of the heart-muscle, large pericardial exudation with hindering of the heart's action, considerable emphysema of the lungs with excessive damming of the smaller circulation), and in the rare case of compression of a large venous trunk just before it enters the right auricle (tumors of the mediastinum).

Local venous stasis is caused by closure or marked narrowing of a more or less large venous trunk. This closure may be produced by compression or by thrombosis of the vein (compression of the cava or the extremity of a venous trunk by tumors); by compression of the cava inferior in connection with the common iliac artery by very large effusion in the peritoneum or by tumors; by atrophic thrombosis of a vein of the extremity, especially the femoral. Not infrequently the collateral veins of the skin take up the conveyance of the blood of the venous stasis; they then become enlarged and sometimes tortuous.[1]

For the cyanosis produced by certain poisons, see Examination of the Blood.

4. The Yellow Skin, Icterus, Jaundice.—The jaundiced state of the skin exists in well-marked cases, with slight differences, almost equally over the whole surface of the body. It is found especially in the conjunctiva, and in slight cases exclusively there and in the other mucous membranes, if the observer will render the spot anemic by pressure (best done by means of a microscopic slide pressed upon the everted lip or upon the tongue). According to the intensity of the jaundice, the tissues are but slightly tinged with yellow or citron color or yellow-green. Only in very severe cases (melas-icterus) does the skin become green or brownish-yellow.

Jaundice cannot be detected by the ordinary means of illumination, since the yellow artificial light does not enable one to distinguish between white and yellow. In slight cases it will first be detected in the conjunctiva. But this must not be confounded with the yellow fat that sometimes exists there, especially in elderly people. In persons with yellow or brown skin the jaundice is revealed by an examination of the mucous membrane.

The yellow color of the skin after taking picric acid or santonin has no relation to jaundice. We distinguish this condition from jaundice by analysis of the urine (*q. v.*) and by determining the etiology of the former.

The icterus of the surface of the body which can be clinically demonstrated is the partial evidence of the distribution of the yellow coloring matter through the whole organism with the exception of a few tissues. It is caused almost without exception by the presence of biliary pigment in the blood. Biliary pigment, however, seems to be formed exclusively in the liver. At least we know, according to the researches of Naunyn and Minkowski, that this is the case in geese and ducks, and there is no reason to suppose that it is different in man. The icterus caused by biliary pigment points, therefore, always to abnormal processes in the liver, and these processes are of such a kind that they culminate in a transference of biliary constituents into the blood.

We may say that the cases in which we observe icterus, with very few exceptions to which we will revert later, are divided into two large groups: either we have to do with purely mechanical, so-called icterus of stagnation (hepatogenous icterus) or with the hemogenous, better hemo-hepatogenous, icterus.

1. The so-called *hepatogenous icterus*, icterus of stagnation, is the

[1] *Vide* Examination of the Veins.

result of an interference with the flow of bile from the liver by an obstruction in the large or in many small biliary ducts, or at the place of entrance of the ductus choledochus into the intestines. This produces stagnation of the bile in the liver and the transference of it into the blood. The causes of this most frequent form of icterus are—(gastro-) duodenal catarrh, with catarrhal swelling of the mucosa and accumulation of mucus in the ductus choledochus; tumors which press upon the duodenal orifice of the ductus choledochus, and especially cancer of the head of the pancreas; ascarides or round-worms (*q. v.*) which enter the ductus choledochus; and also gall-stones which lodge there.

There may be compression of the hepatic duct or of the large gall-duct at the entrance of the liver by tumors (carcinoma, echinococcus) or by scars or by closure of the same by gall-stones. Closure of many small bile-ducts may be caused by so-called intrahepatic gall-stones; possibly also compression of these by marked damming in the branches of the veins of the liver from general venous stasis; finally, catarrh of the smallest bile-ducts may possibly cause bile-stasis and jaundice, as in phosphorus-poisoning.

One consequence of the presence of biliary constituents in the blood is that they make their appearance in the urine. Therefore, the urine in icterus caused by stagnation, with the exception of very light cases, always contains demonstrable qualities of biliary pigment, and sometimes also of bile acids.

In case the flow of bile is much hindered or is wholly stopped, then, partly from the want of bile and partly from the fatty contents, the stools become light, perhaps entirely white or gray-white.[1]

In some cases of severe jaundice there may be still other appearances—itching, various skin affections, minute cutaneous hemorrhages, slowing of pulse, or simple nervous manifestations. In very severe, long-standing jaundice there may be marked heart-disturbances, hemorrhagic diathesis may develop, or, finally, there may arise severe nervous manifestations (cholemia, cholemic manifestations).

2. *Hemo-hepatogenous Icterus.*—It has been long known that icterus occurs in certain cases of poisoning and in certain infectious diseases. These conditions have the common feature that they are accompanied by a marked alteration of the blood, which is chiefly characterized by a dissolution of the red corpuscles and by hemoglobinemia. Formerly it was supposed to be most probable that in such cases hemoglobin-hematoidin, which is identical with bilirubin, was formed in the blood from hemoglobin, and that, consequently, there was produced a genuine blood-icterus. We have, however, mentioned above that in certain animals, and probably also in man, the liver alone is to be regarded as the place of formation of bilirubin. It has farther been demonstrated by Afanassiew and Stadelmann that in cases of dissolution of the blood by poisons a great amount of bile is secreted which is very rich in biliary pigment and is very thick. As a consequence, the biliary ducts are insufficient to carry off this bile, and thus there is a secondary stagnation of bile in the liver. It is therefore to be sup-

[1] The particulars of this condition of the stools and of the urine in jaundice are explained in the chapters devoted to these subjects.

posed that in primary alterations of the blood icterus is produced by stagnation of bile in the liver.

The form of icterus which is dependent on a dissolution of the blood is seen in certain poisons: by chloroform, ether, chloral, chlorate of potash, solution of arsenic, toluylendiamin; in certain infectious diseases, etc., pyemia, yellow fever, sometimes in pneumonia. Here, however, it is necessary to add that probably complicating disease of the liver may have some share.

In primary icterus of stagnation we expect to find biliary pigment in the urine, and this is usually the case, but not always. Here is a weak point in the doctrine of hemo-hepatogenous icterus. However, we must not overlook the fact that in this form of icterus the feces are not discolored, because there is a deficiency of bile in the intestines.

We will not omit to emphasize the fact that icterus does not always follow dissolution of the red corpuscles, with formation of hemoglobinemia. Slight hemoglobinemia—for instance, in slight cases of poisoning, in moderate burns—produces neither icterus nor alterations in the urine; and even more severe cases of hemoglobinemia may cause hemoglobinuria without icterus. On the contrary, only in the most severe cases of hemoglobinemia is there such a degree of polycholia or inspissation of bile as to cause icterus.

Icterus Neonatorum.—We understand by this a very benign variety of jaundice which appears in a considerable number of new-born babies during the first days of life. The explanation of this disease, however, is doubtful. Purely mechanical conditions (the sudden decrease of pressure in the vena porta—Frerichs), as well as processes in the blood (Hofmeier), and other things, have been indicated as causes of the condition; but none of these explanations has been fortified by exact proofs.

There are still other cases of icterus which cannot be included in the two great categories mentioned above. First of all are cases in which no biliary coloring matter can be found in the urine. In most of these cases the color of the skin is not definitely icteric, but only slightly yellowish, often dirty yellowish, much resembling the skin sometimes seen in chronic diseases of the liver, especially in alcoholic cirrhosis and in persons suffering from heart-disease, but also in acute infectious diseases. In the last-named diseases particularly there occurs severe icterus without icteric urine; for instance, in pyemia. From this it may be supposed that jaundice may originate from something else than bilirubin in the tissues.

Urobilin-icterus.—In a minority of the cases just mentioned there is found in the urine, instead of bilirubin, great quantities of a substance related to it, hydrobilirubin or urobilin, which substance is formed from bilirubin by reduction with sodium amalgam, but also under the influence of the bacteria of decomposition; and this substance is also found in the urine during resorption of extravasated blood.[1] It has been believed that in the cases of icterus mentioned above, where no biliary pigment, but urobilin, appeared in the urine, the urobilin was the cause of the icterus (urobilin-icterus—Gerhardt and v. Jaksch). But F. Müller has lately adduced important reasons against the existence

[1] See chapter on Urine.

of an icterus caused by urobilin, which reasons we cannot here discuss.[1]

D. Gerhardt[2] in a series of cases of different kinds of icterus, some of them of the slightest degree, always found bilirubin in the blood-serum and in the tissue-lymph. From this we are to infer that, contrary to what has been said above, icterus would always be caused by biliary pigment. Whether this view will be confirmed is still doubtful.

It is uncertain where urobilin is formed. Tissier recently asserted that it is principally formed in the liver. Healthy liver-cells produce bilirubin out of hemoglobin; diseased liver-cells or those damaged in any manner, however, produce urobilin or certain substances intermediate between these two. Severe urobilinuria only makes its appearance in cases of chronic disease of the liver if there exists an increased dissolution of red corpuscles. On the other hand, F. Müller thinks that urobilin is formed in the intestines. He says it is formed by the bacteria of putrefaction by reduction from bilirubin. He did not find it in the feces and the urine when no bile entered the intestines, and likewise as long as there was no putrefaction in the intestines, as in the new-born. But it is not feasible to discuss this interesting question more in detail here.

5. The Bronze Skin.—Unlike cyanosis and jaundice, this is a condition pertaining only to the skin and mucous membrane. We speak of the chief symptom instead of the true anatomical seat of the disease—viz. the suprarenal capsule, the so-called Addison's disease. It is regarded as a disease of the suprarenal capsule, more frequently tubercular. Connected with it is a degeneration of the ganglia and of the ramifications of the sympathetic nervous system. [The association of this peculiar brown discoloration of the skin is not constant in Addison's disease. It is not so constant in cancerous, but is more common with cheesy, degeneration. The latter condition may be present without bronzing of the skin. On the other hand, the skin may be bronzed, just as "in Addison's disease, without the existence of cheesy degeneration or any other change in the suprarenal capsules. These facts have induced many observers to attribute the cutaneous discoloration rather to changes in the neighboring sympathetic nerves —the solar plexus and the semilunar ganglia."]

The bronze skin is characterized by a brown, gray to black discoloration, especially of the face and hands. There is also the common normal pigmentation of the skin in spots. The discoloration may gradually extend over the whole surface of the body, only the nails and cornea remaining clear.

It is very important to notice that the same discoloration appears upon the mucous membrane of the mouth, and more rarely upon the lips, as very sharply circumscribed, frequently quite small, brown specks.

The discoloration is caused by deposit of pigment in the rete Malpighii. Of course pressure with the finger does not at all diminish it.

Arsenical Melanosis.—When arsenic has been administered for a long time, sometimes even though the doses be small, there is produced a discoloration of the skin, and likewise of the mucous mem-

[1] See chapter on Urine.

[2] *Diss.*, Berlin, 1889.

brane of the mouth, which in every particular resembles Addison's bronze skin. This condition is called arsenical melanosis. After the arsenic has been discontinued the discoloration only imperfectly disappears or it may persist.

6. The Gray Skin of Silver Deposit.—After long-continued administration of nitrate of silver there may be deposits, in certain organs, of very fine black particles (metallic silver or silver albuminate ?), as in the kidneys, intestine, and also in the skin, and especially in the corium, the tunica propria of the sweat-glands.

The skin of such persons, especially of the face and hands, is *gray* or *blackish*. The color is not changed by pressure. In severe cases we also observe corresponding gray specks in the mucous membrane of the mouth.

In a strict sense this is not a diseased condition: the function of the organs which are impregnated with silver do not seem to be in the least disturbed, and these people are perfectly well.

D. Other Pathological Appearances of the Skin of General Diagnostic Value.

1. Acute Exanthematous Diseases.—In some acute infectious diseases a characteristic eruption of the skin has so marked an appearance that these diseases are designated as "acute exanthemata." They are—scarlet fever, measles, German measles, small-pox, and varicella. Here we may pass over the cutaneous diseases which belong here, since they are closely connected with the complete description as these diseases are taught at the bedside.

On the other hand, there are certain other acute exanthematous diseases, less striking, but at the same time of great diagnostic importance. We may here briefly mention—

(*a*) **Roseola.**—This presents a small, round, rose-red, slightly elevated spot. It is generally scattered, is found most frequently upon the abdomen and lower part of the back, more rarely upon the breast and extremities in *typhoid fever*. It appears about the beginning, and generally fades at the end, of the second week. Now and then secondary roseolar spots appear later, which are connected with exacerbations of the disease (involving new portions of the intestine ?).

Secondly, it appears in the form of somewhat larger spots and in the great majority of cases of *typhus fever*. But, except in light cases, it is in this disease petechial—*i. e.* the location of small hemorrhages, which are slowly absorbed. In the beginning of the disease it is often not easy to distinguish this roseola from the eruption of measles.

Further, roseolar spots exist in some cases of acute miliary tuberculosis, and finally in animal poisoning.

Finally, it is necessary to refer in this place to *roseola syphilitica* and to an eruption which appears in the form of small, flat (not raised) spots—*erythema exudativum multiforme*.

(*b*) **Herpes Facialis.**—This consists of a group of small vesicles upon a slightly red base. The vesicles contain at first clear water, then are cloudy, then yellow from pus contained in them. They may be confluent. After a few days they dry up and scale. Most frequently

this exanthem is found in the neighborhood of the mouth—*herpes labialis;* or of the nose—*herpes nasalis;* it may also appear upon the cheeks or the ear.

It makes its appearance at the beginning of some acute diseases, and seems to be especially peculiar to very rapidly rising fever. Above all, it accompanies croupous pneumonia, then epidemic cerebro-spinal meningitis, in which disease it is often quite extensive, finally, sometimes in angina (*angina herpetica*), and a light febrile disease named, in consequence, febris herpetica.

An herpetic eruption also sometimes accompanies the development of *intermittent fever* and the *chill of pyemia.*

(*c*) **Miliaria, Sudamina.**—These are small, remarkably clear vesicles, which reflect the light strongly. Generally they occur in large numbers, especially upon the abdomen. They appear if a patient, after long-continued anhidrosis, begins to sweat profusely, especially in acute, but also sometimes in chronic, diseases. They contain perspiration, and hence the drop which appears after they have been punctured reddens blue litmus-paper.

Still other exanthemata of diagnostic importance could be mentioned here, as the rare scarlet redness in the beginning of *typhoid fever*, the different eruptions of *sepsis*, *pyemia*, and other diseases.

2. Exanthemata from Poisons and the Use of Medicines.—These are of varied character, since they sometimes resemble those of acute diseases—viz. scarlet fever, measles, etc. They may, therefore, easily cause an error in diagnosis. The medicines or poisons which most frequently produce exanthems are—quinin, antipyrin, salicylic acid, opium, morphin, atropin, strychnin, balsams, particularly balsam of copaiba, iodin, bromin (and substances applied locally, vesicants, as mustard). The particulars regarding them belong to works on diseases of the skin, and also to pharmacology and toxicology.

3. Hemorrhages in the Skin.—They arise, it appears, chiefly by diapedesis, and seldom only by rupture of blood-vessels (*rhexis*), and take place by preference, but not exclusively, in dependent parts, especially the lower extremities. They may be of any size—from the smallest perceivable point to the size of the palm of the hand or even larger. The small, punctiform hemorrhages, ecchymoses or petechiæ, are most apt to appear at the hair-follicles. When the hemorrhages are fresh the color is like venous blood. During absorption they are brown-red, later becoming bright brown.

A hemorrhage is distinguished from a circumscribed inflammatory redness of skin *in that it does not disappear upon pressure.* (The small ecchymoses in the hair-follicles, mentioned above, are easily confounded with the latter, especially in cyanosis; further, petechiæ in parts previously inflamed, as in measles, are easily overlooked.)

Simplest test: Press a piece of glass, a microscope slide, upon the suspected spot. A hemorrhage is rendered more distinct, while the surrounding part becomes anemic; an inflammatory hyperemia, on the other hand, disappears.

Hemorrhages take place—

1. *As evidences of a marked hemorrhagic diathesis.* They are then generally extensive in the skin, and, moreover, occur in connection

with hemorrhages from internal organs. They occur in scorbutus, purpura hæmorrhagica; in severe acute infectious diseases, especially pyemia, small-pox, and scarlet fever; in acute phosphorus-poisoning and acute yellow atrophy of the liver; and in all severe cachexiæ.

2. *Without internal hemorrhages*, as a condition limited to the skin: in peliosis rheumatica [*i. e.* purpura occurring with severe pain in the extremities]; also as small petechiæ; almost constantly in typhus fever,[1] often in measles and scarlet fever; moreover, on the legs when the convalescent patient first stands up, especially after typhoid fever; and in badly nourished persons where they have been bitten by pediculi.

3. *In marked venous stasis*, local as well as general.[2] Here belong those punctiform extravasations of blood which are occasionally seen on the face, particularly on the temples, after severe epileptic and eclamptic convulsions and after severe attacks of whooping-cough. More frequently occur here extravasations of blood in the conjunctiva.

4. *As traumatic hemorrhages in and under the skin.* They are sometimes of importance for determining the occurrence of an injury, especially upon the skull.

4. Scars.—These are often important marks for limiting or explaining the clinical history, which, by reason of the scars, can be confined to past local or general diseases or to injuries received.

Thus come under consideration "pock" (small-pox) marks and the scars which may remain after the different *scrofulous* and *syphilitic* diseases of the skin and deeper organs, especially the bones and glands. In internal medicine *scars from injuries* have importance in many nervous diseases (injuries upon the head, the spine, in the course of peripheral nerves).

Here also belong the *scars of pregnancy—striæ* upon the lower part of the abdomen and the upper part of the thigh. Exactly the same scars occur in marked edema,[3] and also sometimes in very fat persons.

5. Ectasia Venarum.—*Varices.*—Visible nets of veins properly belong here, but they will be described in the section on "The Circulatory Apparatus."

E. Edema of the Skin and Subcutaneous Cellular Tissue (Edema, Anasarca).

By these terms we designate an abnormal, marked saturation of the tissues with fluid, which fluid remains wholly or in part distributed in the cellular meshes and lymph-spaces of the tissues, instead of a corresponding quantity of fluid existing in bulk, as its transudation takes place from the blood-vessels to be removed by the lymph-current.

Edema is recognized by puffiness of the skin causing increase of volume of the affected part, and hence, also, the normal outline on the limbs and the trunk are obliterated in consequence of the filling up of the depressions and cavities, and, moreover, there is a tendency to an equal roundness. The skin is smooth, generally slightly shining, and hence, when the edema is marked, very pale in consequence of the

[1] See Roseola. [2] See Cyanosis. [3] See the following section.

diminished circulation. It is very noticeable that the edematous tissue loses its elasticity, so that a depression made by the point of the finger remains for a certain time, sometimes for hours.

Where there is dropsy or a dropsical disposition of the whole body, as in heart and kidney diseases and severe anemias, we generally observe the anasarca first in the most dependent parts of the body, and later these are most affected. It also is most marked where the skin is most loosely attached, having loose connective tissues beneath it.

Hence, in those persons who walk and stand it appears first at the ankles or on the dorsum of the feet (not on the soles and toes, since here the skin is too thick or closely attached); in bed-ridden patients on the inner side of the thigh or in the scrotum and penis, where it is often enormous; on the lower part of the back; sometimes, first of all, in the loose cellular tissue beneath the lower eyelid. One must examine all of these points if he would detect the first evidences of edema.

In very marked cases the deeper parts, especially the muscles, become edematous; the legs may then attain enormous proportions. Moreover, in marked general dropsy there are fluid accumulations in the cavities of the body, giving rise to hydroperitoneum or hydrops ascites, hydrothorax, hydropericardium.

In long-continued edema the skin of the legs and, exceptionally, the lower part of the abdomen may become thickened, as in elephantiasis.

We generally recognize three causes for dropsy of the skin:

1. Venous stasis (hydrops mechanicus).
2. Altered condition of the blood, particularly its becoming watery.
3. Inflammations.

Hence, these following corresponding diseases cause edema:

1. All diseases, local or general, which hinder the return of venous blood to the right side of the heart, as those that have been already mentioned under "Cyanosis."[1]

In local stasis the edema is naturally confined to the roots of the corresponding veins; for example, thrombosis of the right crural vein causes dropsy of the right leg, or compression of the vena cava inferior by an abdominal tumor causes dropsy of both lower extremities.

2. All forms of hydremia (anemia), acute and chronic nephritis, in which the diminished excretion of water, on the one side, and on the other the loss of albumin from the blood, consequent upon the albuminuria[2] occasions the hydremia which is the chief factor in the condition which permits frequent and often marked edema. Yet the hydremia does not always explain the existence of the edema (Cohnheim and Lichtheim).[3]

All other kinds of anemia, hydremia,[4] come under this head when they appear as diseases of the blood or of the blood-making organs, and are secondary to the appearance of wasting diseases and severe acute diseases. Here we frequently see edema of the ankles in patients suffering from chlorosis or from cancer, also when the convalescent patient first stands up.

[1] See p. 38. [2] Which see. [3] See under Albuminuria. [4] See Blood.

The anemia caused by long-continued slight hemorrhages (as those occurring in ankylostomo-anemia) may also lead to moderate edema, for here also we have hydremia, in that the loss of blood is replaced by water in the blood.

3. Edema, sometimes of considerable extent, occurs *in the neighborhood of inflammation* ("inflammatory edema," "collateral edema"). This may be of great diagnostic importance, since it sometimes reveals a deep-seated inflammation.

This is of more interest to the surgeon. To the physician it is important, for instance, in pleuritis with edema of the chest-wall. It shows, with tolerable certainty, that the pleuritis is purulent. Deep muscular abscesses in severe diseases, as in typhoid fever, may easily be overlooked, and may first be recognized by the appearance of edema in the neighborhood, as along the femur, or the lumbar and gluteal regions.

The edema in these different, but so heterogeneous, cases does not have a uniform character: that from stasis is sometimes soft, sometimes very elastic, the latter especially (in marked stasis) exists in the extremities, when it is often difficult, and sometimes impossible, to leave the mark of the pressure with the finger; moreover, in cases of nephritis with a small quantity of urine and marked albuminuria it is sometimes very considerable, but now and then softer. In the different anemias the edema is mostly slight—a scarcely noticeable puffiness. Slight edema disappears between morning and evening or evening and morning, according to the change of position of the body.

Some differences as regards the localization of the edema can be recognized. In patients suffering from heart-disease, for instance, there is a decided tendency to have edema affect the most dependent parts, as the lower limbs, and also the parts of the body most distant from the heart, since the circulation suffers there sooner than elsewhere. Both of these conditions are fulfilled in the dependent feet and hands. But in nephritis the influence of gravity frequently cannot be well recognized: for instance, the edema appears only in the eyelids. Again, the slighter edemas in acute nephritis and also in chronic interstitial nephritis (Bright's disease) are strikingly transient. It is also to be observed that in severe and long-continued acute nephritis, as well as in all forms of chronic nephritis, if the heart-power is weakened we have cardiac dropsy.

The question, Why does edema result from venous stasis, hydremia, or inflammation? has not in all respects been satisfactorily answered. Until recently it seemed to be proved that this is entirely to be ascribed in these three conditions to an injury of the endothelium of the vessels, and by this means occasioned increased transudation into the tissues (Cohnheim). Later, the view has been advanced, and it seems to me has become well established, that the loss of elasticity and the diminished squeezing-out of lymph from the tissues by their being relaxed plays an important, perhaps a chief, part in causing edema (Landerer). This relaxation of the tissues might be caused by the stasis from the increased transudation, or by the hydremia from the deficient nourishment of the tissues by the morbidly thin blood, or, finally, it might be caused by inflammation excited in the neighborhood.

In conclusion, we must not omit to mention that, in rare cases, edemas occur which do not come under the above-mentioned heads—which, moreover, are not accompanied by any other morbid disturbances. Here belong the essential edema of children and the edema of the feet after forced marches.

F. Emphysema of the Skin.

By emphysema of the skin is understood the entrance of air into the cellular tissue. It may be limited to one region of the body, as the neck or the upper part of the chest or the upper part of the abdomen; but it may be spread over almost the whole of the body. It is, in general, a very rare condition.

We recognize emphysema of the skin by the marked paleness over a region which is decidedly elevated above its surroundings. Indeed, on account of the loose fixation of the skin in certain parts, even depressions, as that over the clavicle, or the axillary space, or the intercostal spaces, *may be filled up*, so that thus sometimes on a first glance at the part it seems like marked edema. Sometimes at such places there may even be an elevation of the skin like a pillow. Upon palpation we find that the part is very yielding, like a soft pillow. Quite unlike edema, however, the depression made by pressure immediately disappears. Moreover, upon palpating the part we feel and hear an unusually fine crackling.

The so-called *aspiration*[1] *emphysema* of the skin does not here concern us. It arises from decomposition of a blood-extravasation or abscesses with formation of putrid gases.

The so-called emphysema of skin from aspiration arises from the entrance of air or gas into the subcutaneous tissue, either from without through a wound of the skin or from within from an organ containing air or gas.

(*a*) The entrance of air from without after a wound of the skin belongs to surgery. This occurrence is especially observed in wounds of the neck (tracheotomy), of the breast, in the lower part of the face, and even in wounds of the mucous membrane of the mouth. The wounds in question are sometimes remarkably small.

(*b*) Of much greater interest in itself, as well as from a diagnostic point of view, is emphysema from air or gas entering the cellular tissue from within. Under all circumstances it is occasioned by the rupture, either spontaneously or traumatically, of the wall of an organ containing air or gas. Hence emphysema of the skin may arise—

1. *From any portion of the respiratory tract, from the larynx down.* Deep-seated ulceration of the larynx or trachea may invade the walls of these organs, and thus the air may escape and enter the subcutaneous cellular tissue.

Cavities of the lungs (after previous repeated adhesions between the pulmonary and parietal pleura) may ulcerate into the chest-wall, until, finally, communication with the cellular tissue is established. Then the

[1] The name "emphysema" is not quite accurate, since generally the air is driven in under pressure, as is shown by what follows.

pressure of a severe paroxysm of cough may cause the air in large quantity to spread out quickly under the skin.

Single pulmonary alveoli may burst from any very high intrathoracic pressure, as severe cough, especially in children with whooping-cough, bronchitis, or emphysema; sharp crying; severe exertion, as blowing on wind instruments, or women in childbirth; and air may enter under the pleura or into the interalveolar tissue, reach the mediastinum, pass along the mediastinal space into the subcutaneous tissue of the neck, and so spread onward.

Wounds of the lungs (as fracture of the ribs without external wound) may either directly cause emphysema of skin, or, passing the mediastinum as above, take the same course.

2. *From the esophagus, stomach, or intestines, and, indeed, from the esophagus again through the mediastinum:* from the stomach or intestines by adhesions with the abdominal wall and invasion of the cellular tissue there; from traumatic rupture of the esophagus, more frequently from ulceration, especially in connection with carcinoma of the esophagus; with any kind of deep seated ulcerations of the stomach and bowels.

Sometimes there occurs extensive decomposition of the cellular tissue, especially if emphysema of the skin is produced by gases from the intestinal canal (mixed with intestinal contents). Very often, however, the emphysema remains without such action. It may then spontaneously disappear. But, at the same time, the emphysema is generally a final development, partly on account of the severity of the primary disease, and partly because it causes severe dyspnea, as, for instance, that in the mediastinum, and hence is a very serious condition.

From a diagnostic point of view emphysema of the skin is of great importance, since it affords a conclusion regarding the diseases mentioned. Under some circumstances it may afford the first and only symptom, as in the affections of the esophagus.

V. THE TEMPERATURE OF THE BODY. FEVER.

It is a well-known peculiarity of warm-blooded animals, if the organization is otherwise sound, that with remarkable constancy they maintain a certain internal temperature which is subject to very slight variations. If that peculiarity is lost, if the temperature departs from the normal, then, almost without exception, a morbid disturbance is present. A knowledge of this fact, and especially of the elevation of the specific heat in disease, attracted the attention of physicians to the temperature of the body from the earliest time. Recently, however, the measurement of the temperature has become of the greatest diagnostic aid, indispensable for every scientifically educated physician.

1. The Terms Used and the Method of Taking the Temperature.—Judging of the temperature by *laying on of the hands* is under all circumstances deceptive. Great errors cannot be avoided even if covered parts of the body are selected, while uncovered parts cool so rapidly as to furnish no standard.

We measure the temperature with the *Centigrade or Celsius's ther-*

mometer, with the scale divided into tenths, from about 30° to 45°. There is no need for a thermometer with indications below 30°.[1]

In France the Réaumur scale is sometimes used; in England and America the Fahrenheit is generally used. To convert from one standard to another the following formula is used:

$$1° \text{ C.} = \tfrac{4}{5}° \text{ R.} = (\tfrac{9}{5} + 32)° \text{ F.}$$

It is further to be remarked that in Germany still, especially at the public baths, the baths are frequently measured and are prescribed according to the Réaumur standard.

As regards the selection of the instrument, it is to be recommended to use only officially tested thermometers, which are provided with a number and a certificate as to their correctness. The price of them is a little higher than that of those which have not been tested. The guarantee for the correctness of the tested instruments extends, however, only a certain time. As the glass changes a little in the course of time, it is necessary to have them again tested after a few years. Thermometers without a test certificate offer of course less guarantee for correctness. Always before using such a thermometer one should compare it with a so-called normal thermometer. But as normal thermometers are very expensive, officially tested ones are at the present time to be preferred.

When a comparison with a normal thermometer cannot be made, an approximate determination may be made by taking the temperature in the axilla of a healthy person upon, say, six different days an hour after breakfast. A thermometer which is correct in its reading must then give an average reading of 37° C. or a little less (Liebermeister).

Instruments with cylindrical bulbs are to be preferred to those with spherical ones. Thermometers are made so that by a prismatic form of the front surface the column of mercury appears broadened, magnified laterally, which greatly facilitates the reading of the index. Very small thermometers, from different reasons, are all of them unreliable.

Maximal [self-registering] thermometers have a decided advantage, because it is not necessary to read them while the temperature is being taken.

The temperature may be taken in the axilla, the rectum, or in the vagina. Taking the temperature in the mouth and also in freshly passed urine is to be avoided. The rectum and vagina are in reality the only places for an exact determination, because they are closed cavities within the body, and their temperature is uniform with that of the inside of the body. For this reason, and because they enclose the thermometer almost completely, they have also the secondary advantage that in them the mercury rises very quickly to its maximum. In the rectum or vagina most instruments record the temperature of the body within five minutes. But, unfortunately, in a majority of cases, for reasons of delicacy, the taking of the temperature in these places cannot be practised. But if the rectum be chosen, the oiled instrument is introduced to the depth of about five centimeters and left for five minutes. If there happen to be hard feces in the rectum, it is necessary

[1] See below.

to remove these first, because experience has taught that in such a case the record of the temperature is too low.

The axilla is not a perfectly closed cavity, but becomes a closed cavity only after the upper arm is pressed against the chest-wall when it begins to take the temperature of the body, but it almost never completely reaches it. On the contrary, the temperature here always remains a little below that of the inside of the body. In every case this is a few tenths of a degree lower, but in very thin subjects, where the cavity closes imperfectly round the thermometer, it remains much below the temperature of the inside of the body.

The time required for the skin of the axilla and the instrument to reach the maximum temperature varies greatly, especially in collapsed patients with a cool surface of body and if bathed in perspiration. It is therefore necessary always first to wipe the axilla dry. Generally the maximum temperature is reached in ten to fifteen minutes. The best way is to look at the index after eight to ten minutes without moving the thermometer from its position, and to examine it again after ten to twelve minutes, to see whether the mercury is still rising. Care has to be taken to carefully place the bulb in the axillary cavity and to keep the upper arm closely against the chest-wall while the thermometer is in place.

From what has been said, it is evident that the so-called minute thermometers, apart from their inexactness, are of no use for taking the temperature in the axilla, since the axilla itself does not reach the temperature of the body in so short a time.

Generally one has to be content with taking the temperature in the axilla, and, as a rule, no important error is made if he considers the temperature taken in this manner as the temperature of the body. On the other hand, if one measures the temperature in the rectum and axilla alternately, it is well to add in the former case about two-tenths of a degree [two-fifths of a degree Fahrenheit].

From what has been said above, in collapsed and greatly emaciated patients one should occasionally at least make a controlling measurement in the rectum or vagina. In such cases I have not infrequently found that the temperature in the axilla, in spite of the greatest care in taking it, was one degree [Cent.] or more too low, even after the thermometer had been in the axilla a quarter of an hour. But in children and in unruly patients the rectal measurement is always to be preferred. In these cases one has particularly to look out that they remain quiet, lest the thermometer be broken.

[Practical experience teaches that the best place to take the temperature varies in different cases and subjects. In the case of children and infants the rectum is the best place. This is also true of many adults who are violent or restless, and likewise very aged persons. If the patient is conscious and quiet, it is much more convenient and also more reliable during a series of observations to take the temperature by placing the thermometer under the tongue, and directing and assisting the patient to keep the mouth tightly closed, with the instrument steadied in place by grasping it between the teeth. Thermometers of the best make are thoroughly reliable for reading after being in the mouth for three minutes. It is necessary to know whether the patient

has recently been taking any very hot or very cold fluid or ice in the mouth. Apart from this source of error, the mouth is, on the whole, more uniformly reliable for temperature observations than the axilla, whose form and the conditions of the skin as regards moisture, etc., varies so greatly in different subjects.]

If the thermometer is not self-registering, it must, of course, be read before it is removed. After using the thermometer in either the rectum or vagina it must, in every case, especially if there is the least suspicion of an infectious disease, be carefully disinfected. [No matter where the thermometer is used, it ought always to be immediately cleaned most thoroughly.]

A single use of the thermometer may be of great value. But it is still more important, as will be shown below, to follow the state of the temperature progressively and to ascertain its course. For this purpose it is necessary to measure it at stated intervals. How frequently this must be done in order to ascertain the course of the temperature must be determined by the particular disease. The thermometer should be used at least twice in twenty-four hours (at about 8 A. M. and again at about 5 P. M.). In diseases with high fever, according to the rapidity with which the oscillations of the temperature are completed, the thermometer must be used every three hours, every two hours, or even hourly. Where the changes of temperature are very marked it may be of interest to observe it every quarter-hour. It is to be understood that where it is proper to do so the use of the thermometer should, as far as possible, be suspended at night, in order not unnecessarily to disturb the patient's sleep.

The record of the course of the temperature may be indicated by a curve. Charts suitable for this purpose of various kinds are to be had. They serve also for the record of the pulse and respiration. Nowadays, in every case of severe fever the physician ought to prepare such a fever-curve.

In what follows the statements regarding the temperature refer to measurements taken throughout in the axilla:

2. The Normal Temperature of the Body.—The average temperature is 37° C., and varies from this about 1.25°—from 36.25° to 37.5° C.

The variations are of different kinds and have different causes. Of least interest, since they are only very insignificant, are those dependent upon age (in children, except the day after birth, a few tenths higher than later; in old people, again, a little higher); an elevation after meals; an elevation after severe exertion.

But the periodic daily variations are more important. They follow the following course: In early morning, between two and six, the "daily minimum" is reached, and then with considerable (not perfect) regularity it rises to the "daily maximum" between five and eight in the evening. From that point, during the night, it again declines. The difference between the minimum and maximum, the "daily difference," is about 1° C. (in rare cases even nearly 2° C.).

After severe exertion the temperature rises quite a considerable amount higher, especially in the sun (Obernier observed that in the case of a person running it rose to 39.6° C.) and in very warm baths.

3. Elevated Temperature; Fever.—A great number of morbid conditions produce in man a temporary rise of temperature. Most of these diseases are of an infectious nature, but some of them are undoubtedly non-infectious; for instance, certain diseases of the central nervous system, anemias and other conditions, can increase the temperature of the body. It has long been the custom to call every increase of temperature of the body a fever. But at the same time it has not been overlooked that in almost all febrile conditions, apart from the increase of the temperature, there are present still other phenomena which have been recognized as a complex of symptoms of fever. These phenomena are—a general feeling of illness, lassitude, sometimes graver disturbances of the functions of the brain, increased frequency of the pulse and respiration, with increased intake of oxygen and increased discharge of carbonic acid, want of appetite, thirst, disturbance of digestion. Frequently, in higher fever, there is decrease in the amount of urine and a relatively (*i. e.* in proportion to the amount of nourishment taken) increased discharge of the nitrogenous end-products of the metabolism of the body, particularly of urea and uric acid, as a sign of the destruction of the albumin of the body, and in high fever albuminuria. After the fever has continued for a certain time there are loss of weight and emaciation in consequence of the increased metabolism.

The more exactly the febrile diseases have been examined, the more evident it has become that, besides the increase in temperature, to most of them also belong all, or almost all, of the above-mentioned phenomena, and furnish stronger reasons for the opinion that fever is a complex of processes and of symptoms, the only question being to which part of this complex the increase of temperature belongs. At the present day this question seems to be finally decided, contrary to the former opinion, that the increase in temperature is a partial phenomenon of the febrile state, which is co-ordinate with the other phenomena.

It cannot be denied that some of the individual phenomena of the complex of febrile symptoms may occasionally be missing; for example, the acceleration of the pulse may occasionally be absent in the beginning of meningitis, or the acceleration is proportionally less, as in typhoid fever, or occasionally the disturbance of appetite does not take place in chronic febrile conditions, etc. But it is of especial significance that even the increase of temperature may be absent; for example, in pneumonia in old people. But this circumstance ought not to give occasion to doubt that the symptoms of fever generally belong together. As a rule, this complex of symptoms always recurs, at least in infectious diseases, and we ought not to attempt to separate this complex of symptoms. The rise of temperature, therefore, is only a single phenomenon of the febrile state, but is very constant, and therefore often at once determines the judgment of the gravity of the disease. Hence it is well to continue to regard the increase of temperature and fever as having the same value. Only the physician must never forget that he has before him a complex of phenomena, and that exceptionally the increase of temperature may not be present.

The febrile temperature in itself shows a remarkable difference from

the normal temperature: it is more changeable—that is, it is quickly and considerably lowered and also increased by different external and internal influences. This is in marked contrast with a healthy organism, which keeps a constant internal temperature with great tenacity. For instance, the febrile temperature increases if the patient is covered very warmly, if the temperature of the room is high, sometimes also after nourishment has been taken. Psychical influences, as fright and anger, have the same effect. On the other hand, the temperature becomes lower if the temperature of the room is low, markedly so from cool bathing, likewise when there is moderate loss of blood, as with the first menstruation or when there is internal hemorrhage. As regards the latter, the accompanying decrease of temperature can be of important significance in making an early diagnosis; thus in typhoid fever we are to think of an intestinal hemorrhage when there occurs a sudden seemingly spontaneous fall of temperature.

Height and Form of Febrile Increase of Temperature.—With reference to bodily temperature Wunderlich has prepared the following table:

I. Normal temperature, 37° to 37.4° C.
II. Subfebrile temperature, 37.5° to 38° C.
III. Febrile temperature:
 (*a*) Slight fever, 38° to 38.4° C.;
 (*b*) Moderate fever, 38.5° to 39° C. morning, and 39.5° C. evening;[1]
 (*c*) Considerable fever, 39.5° C. morning, and 40.5° C. evening;
 (*d*) High fever, 39.5° C. morning, and 40.5° C. evening.

[*Comparison of Thermometric Scales.*

	Cent.	Fahr.	
	34°	93.2°	
	35	95	
	36	96.8	
Normal temperature,	37	98.6	Normal temperature.
	38	100.4	
	39	102.2	
	40	104	
	41	105.8	
	42	107.6	
	43	109.4.]	

If the temperature reaches 42° C., then we speak of *hyperpyrexia, hyperpyretic fever.* While the higher temperatures even of high fevers do not occasion direct danger to the organization, in hyperpyrexia the temperature is directly dangerous to life; it generally leads to a fatal issue.

In exceptional cases still higher temperatures have occasionally been recorded. For instance, temperature of 45° C. [113° F.] has often been observed. The "record" is probably held by the case reported by Teale where the temperature frequently rose to 50° C.

[1] Regarding this difference between morning and evening temperatures, see under Remission.

[123° F.]. This was a case of lesion of the cervical part of the spine, which recovered. The injury to the spinal marrow was most probably the cause of the increase of temperature.

The course of the temperature in twenty-four hours can vary much only in fever. Most fevers show distinct fluctuations, in that toward morning the temperature falls more or less (*remission*) until it reaches the daily minimum; thence in the course of the day it rises (*exacerbation*), and toward evening reaches the daily maximum. The difference between the daily maximum and the daily minimum in fever is called, as in normal temperature, the daily difference. While the course of the temperature in fever is analogous to that of health, not unfrequently the minimum and maximum come at quite a different time; as, for instance, the maximum may be at midday or at midnight; a complete reverse may even take place, so that the maximum occurs in the morning and the minimum in the evening (*typus inversus*).

From this it is seen how the temperature must be exactly measured every hour of the day and night if it is of importance to know whether a patient has fever or not. There have been cases when the persons were thought to be without fever until the physician thought of ascertaining the temperature at an unusual hour, as at night.

The exacerbation of the fever is frequently connected with shivering. If the temperature rises very rapidly (it may rise several degrees in a single hour), generally there is a chill—that is, a decided feeling of chilliness, with severe shaking of the whole body and chattering of the teeth, where very soon, contrary to the subjective chill, there appears a high internal temperature. The skin is at first pale, livid, and generally cool; toward the end of the chill, however, it is regularly very hot.

On the other hand, the decrease of bodily heat is frequently accompanied by perspiration. A rapid change in temperature seems in itself to be able to produce perspiration; but specific toxical influences probably play a part in certain diseases which are accompanied by frequent and abundant perspiration, as in tuberculosis and [acute] articular rheumatism.

According to the amount of the daily difference we distinguish three types of fever:

Continued fever: daily difference not more than 1° C. (chiefly high temperature).

Remittent fever: daily difference over 1° C.

Intermittent fever: maximum very high, minimum within the normal (or even below).

4. The Subnormal Temperature.—It begins at 36.25° C.; the lowest observed temperature is 22° C.

1. It is observed in febrile diseases as an expression of two directly opposite conditions—namely:

(*a*) In a sudden fall of the high fever with an advance to recovery, the "crisis," the critical decline of the fever. In this case the temperature falls during perspiration sometimes to below 34° C., and only in the course of one, two, or three days again returns to the normal. We recognize the "crisis" by the simultaneous diminution of the frequency of the pulse and the respiration, and the feeling of comfort and returning health by the patient.

(*b*) In the so-called collapse. In this condition there is generally a very rapid fall of the temperature, and at the same time a sudden failure of the heart, with (as is the contrary in "crisis") increase of the frequency of the pulse, with paleness and general failure of strength. The condition of collapse may pass over, when there generally is an immediate rise of temperature again to the former point; or it may pass on to a fatal termination.

On the chart of the fever-curve the line of the falling temperature is crossed by the rising line of the pulse-curve in a characteristic way.[1] Sometimes, in a case of collapse ending fatally, the pulse-line sinks parallel with the temperature-line.[2]

2. It occurs sometimes temporarily in severe hemorrhages, also sometimes, for a short time, in all kinds of chronic diseases, especially in those of the heart and the lungs. If the temperature suddenly falls, accompanied by weakness of the heart and general prostration, then also we speak of collapse.

3. *Continuing subnormal temperature*, extending into a number of weeks, is very rare. It may exist in all severe wasting diseases and in diseases of the brain.

5. Diagnostic Value of the Temperature, especially of its General Course.—If we eliminate an elevation of temperature due to bodily exertion or to being in heated surroundings [as in a hot room or hot bath], an increased temperature of the body is otherwise a certain proof that a morbid state is present, and that it is one of those which produce fever. In this lies the first diagnostic value of a measurement of the temperature. Of this a few examples may be given:

1. Frequently the elevated temperature, with some indistinctive complaints (or, in the case of children, abstinence from food with restlessness), is the only sign of a disease just commencing or of one that has been going on for some time. Ascertaining the temperature is, then, of deciding significance in that it leads to a more careful examination and more extended observation and to directing suitable care of the patient. A high morning temperature generally points directly to an acute infectious disease.

2. In marked cachexia, without distinct organic disease, the existence of temporary fever indicates tuberculosis with considerable probability.

3. A single chill accompanied with a rise of the temperature to about 40° C. may, in a given case—say of a disease which from experience sometimes causes suppuration—lead to the diagnosis of suppuration, as in gall-stones, renal calculi, after injuries to the skull, as brain-abscess; also here belongs puerperal fever, or, under certain circumstances, it may possibly be malaria.

But the continued observation of the course of the temperature is of still greater importance. It advances medical knowledge in various ways:

1. The course of the fever in a number of diseases is so typical that from the temperature alone the diagnosis may often be made with great probability, sometimes with certainty. At any rate, taken in association with other symptoms, it is always an important aid in diagnosis.

[1] See Pulse.

[2] See Pulse.

2. Moreover, during the progress of a febrile disease the temperature not infrequently gives notice, by its unusual behavior, of the

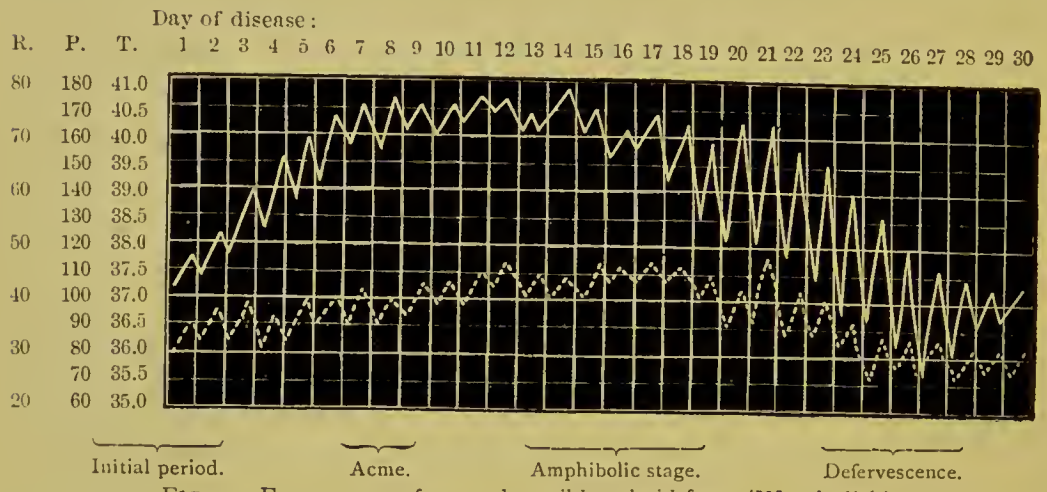


FIG. 1.—Fever-curve of a regular mild typhoid fever (Wunderlich).

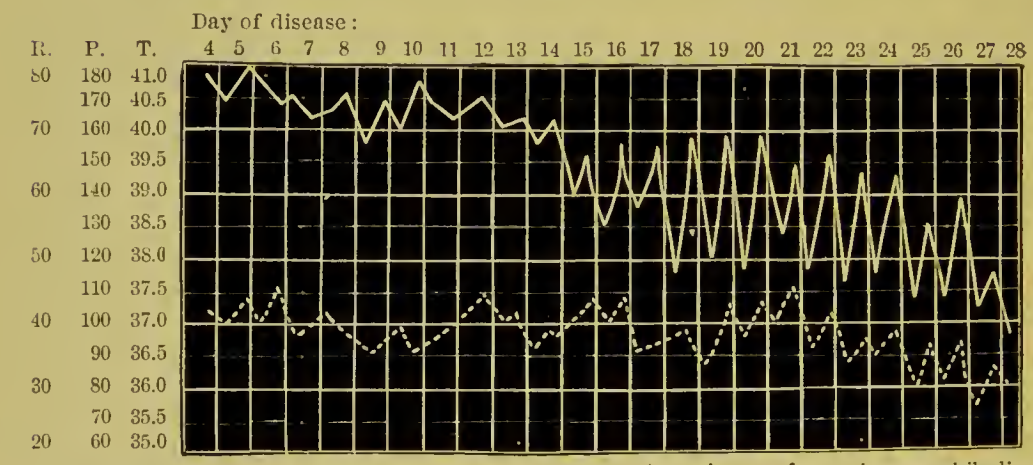


FIG. 2.—Typhoid fever: female, age 23. Fourteen days' continuous fever, then amphibolic stage of relatively longer duration.

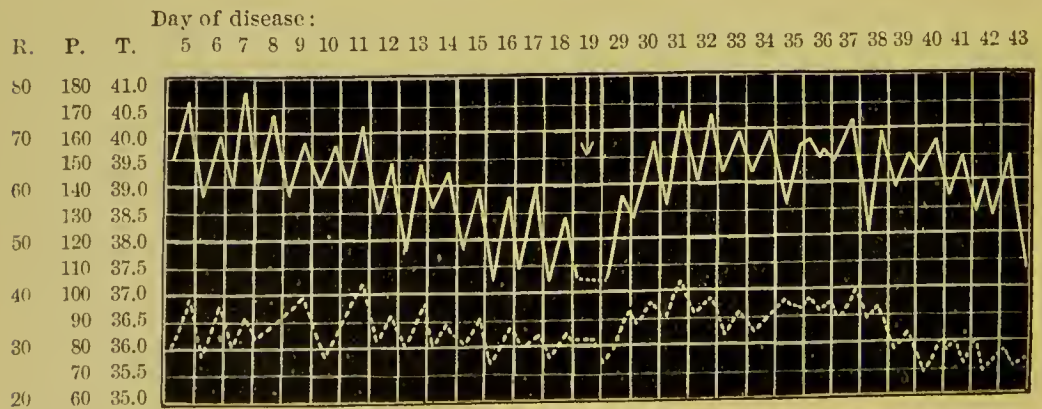


FIG. 3.—Typhoid fever, with recurrence of fever. Servant, 23 years old. The interrupted line (see the arrow) indicates ten days' apyrexia. Rapid defervescence at the end.

occurrence of an unusual event. Hence, not infrequently we first become aware of an exacerbation or of a complication in a given disease by a specially high rise of the temperature. A sudden fall of the

temperature may give notice of collapse, or a change to a fatal issue, or an internal hemorrhage, as of the bowels in typhoid fever.

In the following the most important typical courses of fever are briefly set forth:

1. Continued fever exists especially in two diseases, typhoid fever and croupous pneumonia; also in typhus fever, sometimes in erysipelas

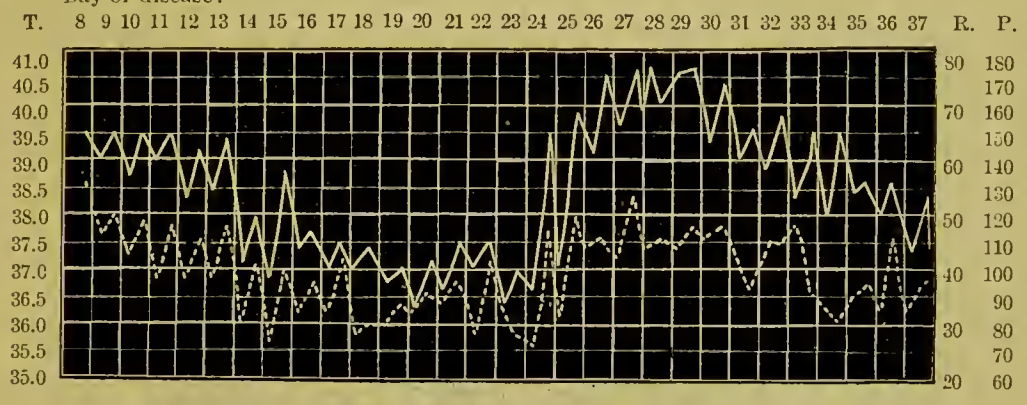


FIG. 4.—Typhoid fever, slight, with tolerably severe recurrence of fever. Maiden, 10 years of age.

and miliary tuberculosis. In a case of severe fever, with the diagnosis doubtful, a fever continued through several days points with probability to typhoid fever, and next to acute miliary tuberculosis.

In abdominal typhus [typhoid fever] the fever rises for several days by equal steps—"initial period;" reaches the summit, at which it remains as a continued fever one, two, or more weeks; then, as a

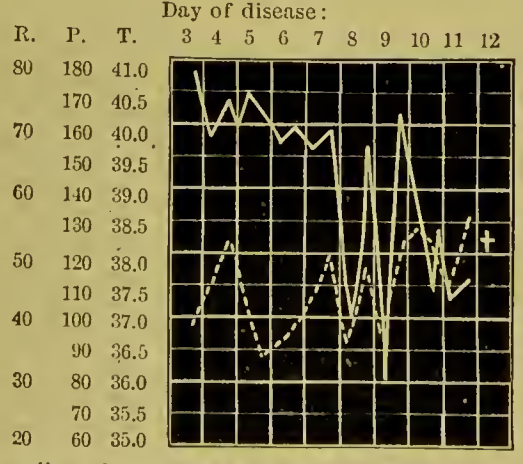


FIG. 5.—Fatal uncomplicated typhoid fever. Temperature and pulse cross each other. Female, age 37.

rule, it gradually becomes a remittent fever of such a character that at first the daily maximum remains high, with the minimum going lower ("the double stage" ["the long-continued paroxysm"]—the minimum may even go below the normal); then the defervescence begins, the maximum declining; this usually reaches the normal in a few days. The remittent and defervescent stages may be protracted for

some time, even as much as a week—"slow typhus." Moreover, the temperature may, after it has somewhat declined, again rise: "recurrence;" or the disease, after the temperature has reached the normal, may begin anew, in the same manner as at first: "renewing" (see regarding these points Figs. 1 and 2).

There are all manner of variations from this behavior of the temperature in typhoid fever, so that a single case seldom really pursues a

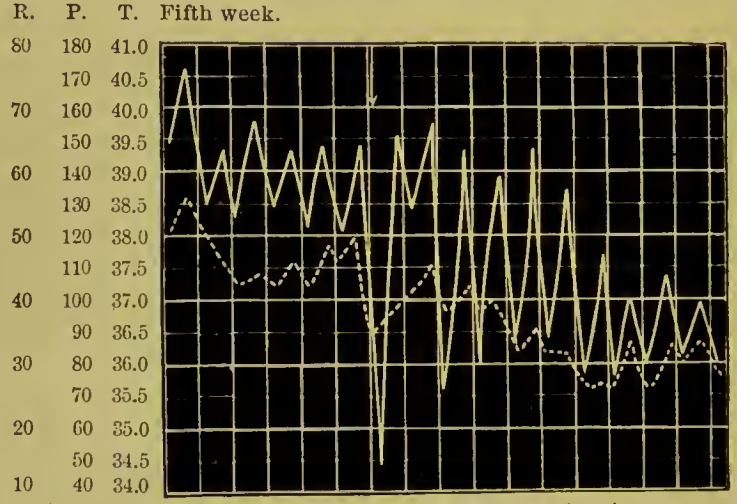


FIG. 6.—Typhoid fever with abortion (see arrow). Age 25.

typical course. Particular variations partly declare themselves by the changeable character of the febrile temperature, which was mentioned on page 55; but the fever-curve will especially be affected by the administration of antipyretics.[1] But, particularly, every exacerbation of

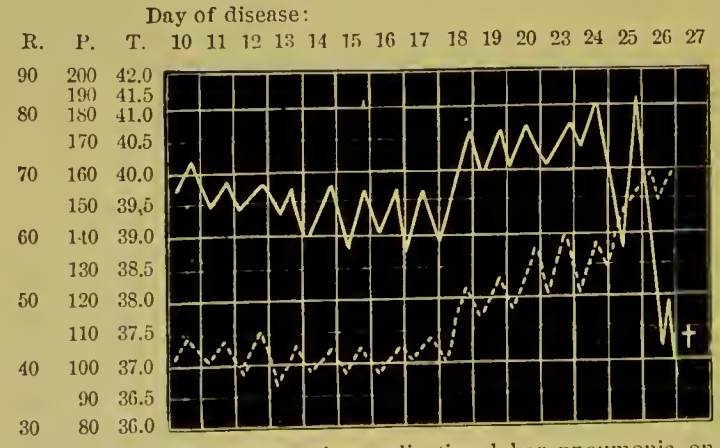


FIG. 7.—Typhoid fever (moderately severe) complicating lobar pneumonia on the eighteenth day. Collapse. Fatal. Female, age 21.

the temperature should cause the physician to think of complications (Fig. 7), and a fall of the temperature, of collapse (Fig. 5), and also of possible intestinal or other loss of blood (Fig. 6).

[1] The antipyretic treatment, especially with internal remedies, has no doubt the result of rendering the course of the fever untypical, and so destroying its diagnostic value. Therefore, until the diagnosis has been established in a case of febrile disease, the internal antipyretic treatment ought, if possible, to be suspended.

In pneumonia (see Figs. 8 to 11) the temperature rises very rapidly ("initial period," lasting a few hours), often accompanied by chill, then remaining as a high continued fever. From this it may decline, also very rapidly—in a few hours—to or below the normal, with a simultaneous decline of the pulse and the respiration, and generally with severe sweating. Or the defervescence may be somewhat slower, occupying one or two days. The former way is called "crisis" (critical sweat), the latter "lysis"; midway between these two is "protracted crisis."

Sometimes the day before the crisis the temperature suddenly falls very rapidly, and then again rises—"pseudo-crisis" (distinguished from collapse by the pulse and the general condition, as referred to under "the subnormal temperature"). Or there is exacerbation of the temperature just before the crisis, rising from, say, 40° to 41° C.—"critical perturbation."

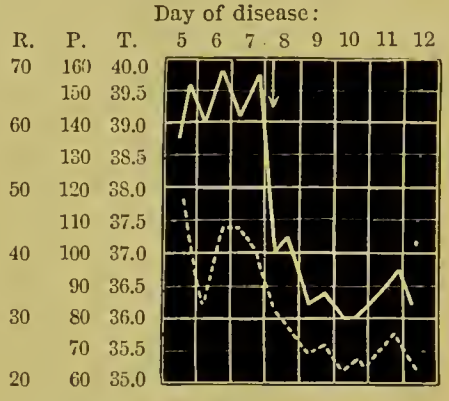


FIG. 8.—Croupous pneumonia, right lower lobe. Male, age 33. Continued fever. Crisis.

2. *Remittent fever* is often met with (Figs. 12 and 13). It may exist some time during the course of any febrile disease. While the temperature of continued fever is generally high—about 40° C.—the fever may remit, whatever its height. If the maxima are low, the minima may easily be normal—a behavior which, strictly speaking, must be

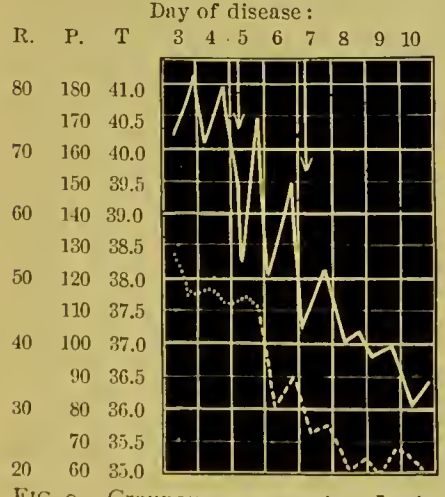


FIG. 9.—Croupous pneumonia. Lysis. Varnisher, age 39.

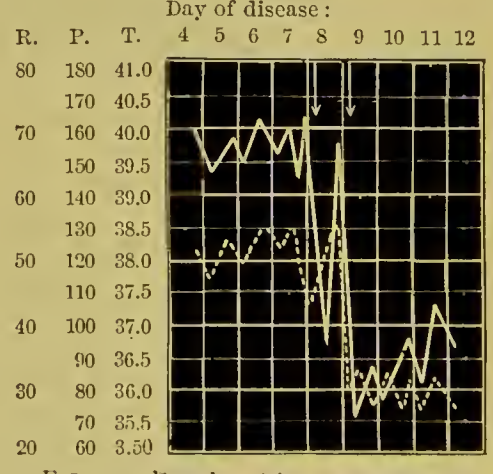


FIG. 10.—Pseudo-crisis in pneumonia. Male, age 25.

considered as intermittent fever. Remittent fever belongs to a great variety of conditions, but especially to chronic tuberculosis.

If the maximal points of the curve are high, the temperature often falls pretty rapidly, accompanied with chills and night-sweats (*hectic fever*). In tuberculosis of the lungs of long duration the continuous line of the evening temperature sometimes describes peculiar low (flat) arcs, which recur with a certain degree of regularity. According to

the observations of C. Turban, these circular lines also sometimes appear when a case of phthisis, which has been accompanied for some

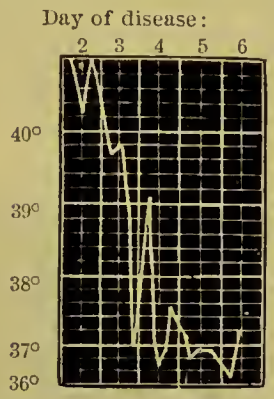


FIG. 11.—Pseudo-crisis and crisis in pneumonia (Wunderlich).

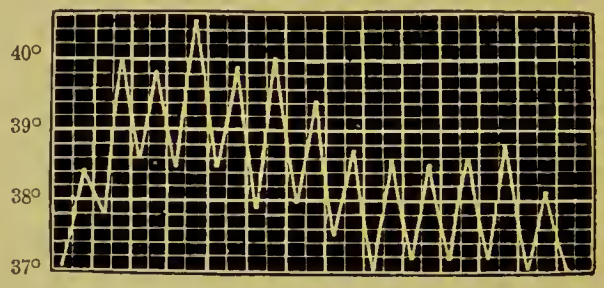


FIG. 12.—Remittent and intermittent fever in catarrhal pneumonia (Wunderlich).

time by fever, is defervescing. The defervescence always takes a long series of weeks (compare Fig. 15). Turban supposes that cases of

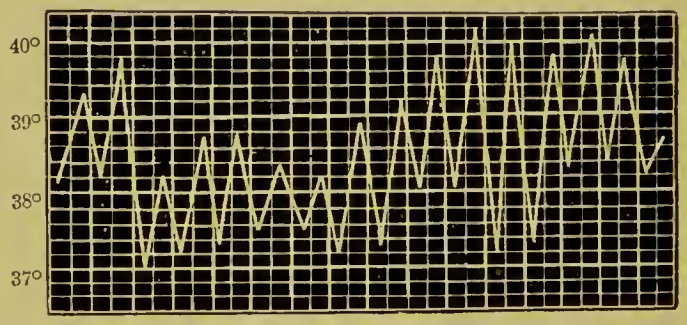


FIG. 13.—Hectic fever in tuberculosis of the lungs.

phthisis which defervesce in circular (arc) lines are connected with an infection by streptococci.

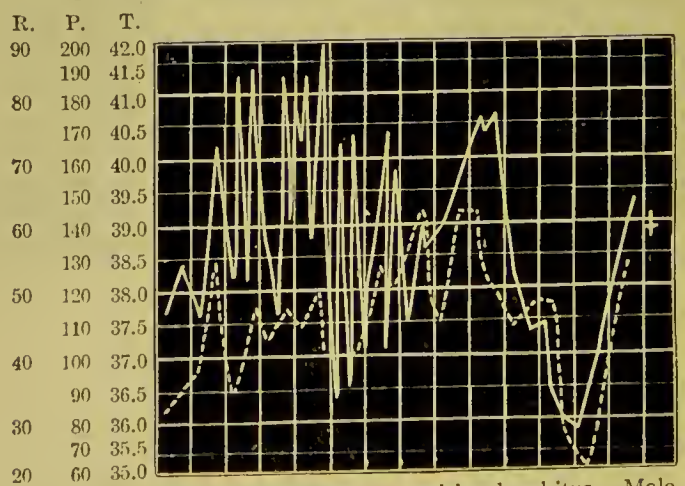


FIG. 14.—Myelitis transversa. Pyemia caused by decubitus. Male, age 32.

3. *Intermittent fever*, in a general sense, occurs in combination with remittent fever (see Fig. 12). The hectic fever mentioned above as

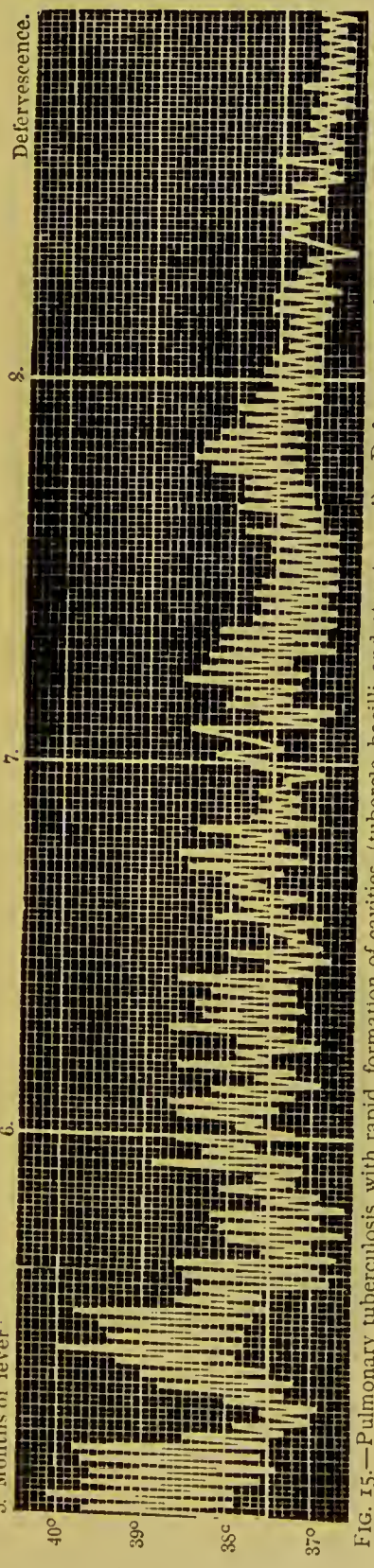


FIG. 15.—Pulmonary tuberculosis, with rapid formation of cavities (tubercle bacilli and streptococci). Defervescence followed by continued good health (observed by Dr. C. Turban).

accompanying remittent typhus [relapsing fever] is often also an intermittent, in which the minimum may even be subnormal.

A peculiar form of intermittent fever is observed in pyemia, where the temperature during chill may rise two, three, or more times in twenty-four hours, and soon fall, with sweat and great exhaustion, then again rising. The pulse is generally very frequent, and the patient often gives the impression, by the great prostration during the sweating stage, of going into collapse; in fact, a condition of collapse sometimes exists with the fall of the temperature (see Figs. 14, 17).

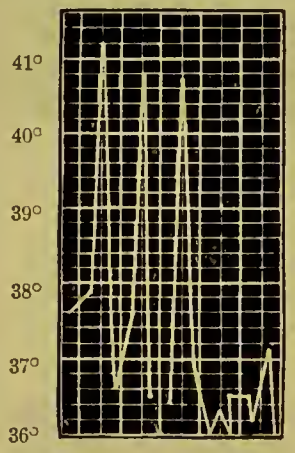


FIG. 16.—Quotidian intermittent fever (Wunderlich).

In a narrower sense, however, we designate as *intermittent fever* the course of temperature of a special form of malaria. In this there is a continual alternation: between-times, without fever (apyrexia); a quick, high rise, and, after a short time again, a rapid fall of temperature (often below normal)—"fever paroxysm." Severe chills and perspiration accompany these attacks of fever. The attacks recur with great regularity, either every twenty-four hours (quotidian), or forty-eight hours (tertian), or seventy-two hours (quartan). Sometimes the attacks recur one or more hours earlier on successive days (anticipating), or they may recur later each time (postponing). In these forms of fever the diagnosis is made certain by the fever-curve (see Figs. 16, 18, 19).

4. *Recurrent fever* (Fig. 20) only exists as a renewal of a febrile disease or a disease known as *relapsing fever*. There is an attack of fever very like that of pneumonia, with sharp transitions and very severe sweating, the temperature falling often to 34° or 35° C., and apyrexia; then a relapse after five to eight days, with a chill, followed

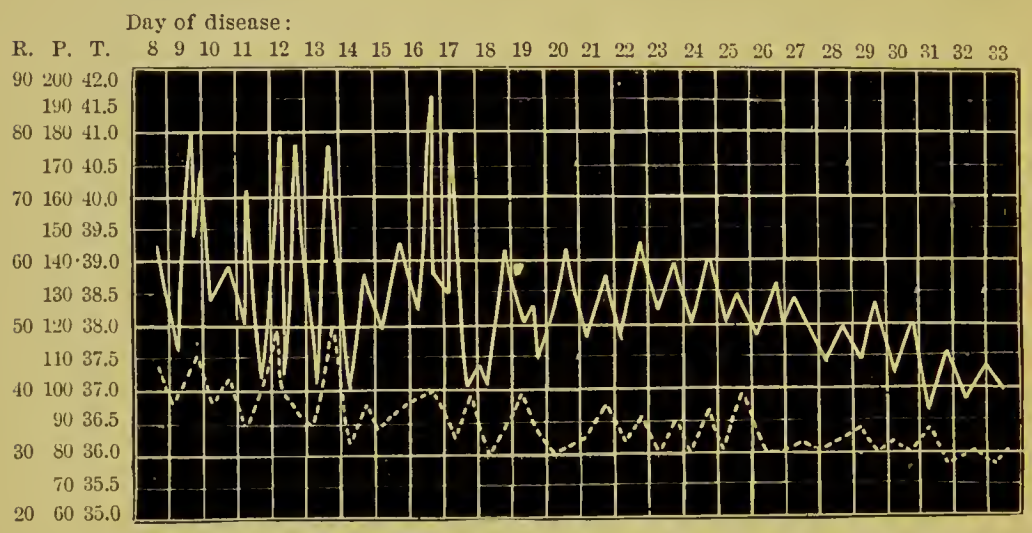


FIG. 17.—Cryptogenetic septico-pyemia, recovery. Female, age 44.

by a high continued fever, which, in turn, ends in five or six days by a critical sweat, new apyrexia, fresh relapse; and so over and over again, but each new attack with less fever and of shorter duration.

5. Not infrequently a quite irregular fever will be met with. Its course is such that sometimes one cannot speak of any daily remission

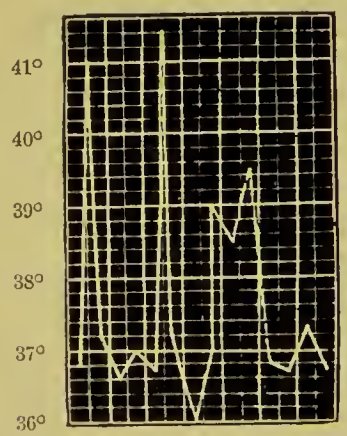


FIG. 18.—Tertian intermittent fever (Wunderlich).

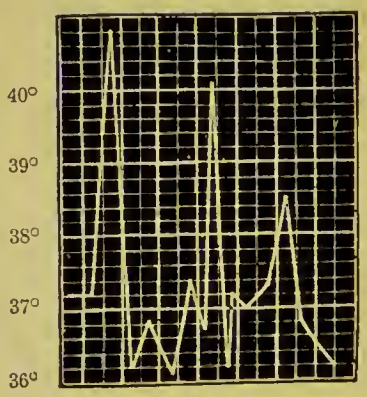


FIG. 19.—Quartan intermittent fever (Wunderlich).

—at least, the lowest daily temperature comes at a variable hour of the day or night. But this fever may be of diagnostic value. In acute meningitis a continuing irregular movement of the temperature speaks against tuberculosis and against ordinary purulent meningitis, but, on the contrary, for epidemic cerebro-spinal meningitis. A pronounced irregular fever in an acute disease generally speaks against any of those diseases which manifest themselves by any typical fever.

6. Local Elevation or Lowering of the Temperature.—**1. Elevation of the Temperature.**—In internal medicine this is seldom of diagnostic aid. We meet it where there is any kind of inflammation which is near the surface, as in surgery. In unilateral pneumonia also a careful measurement shows an elevation of the temperature in the axilla of the affected side. In recent paralysis of any sort the tempera-

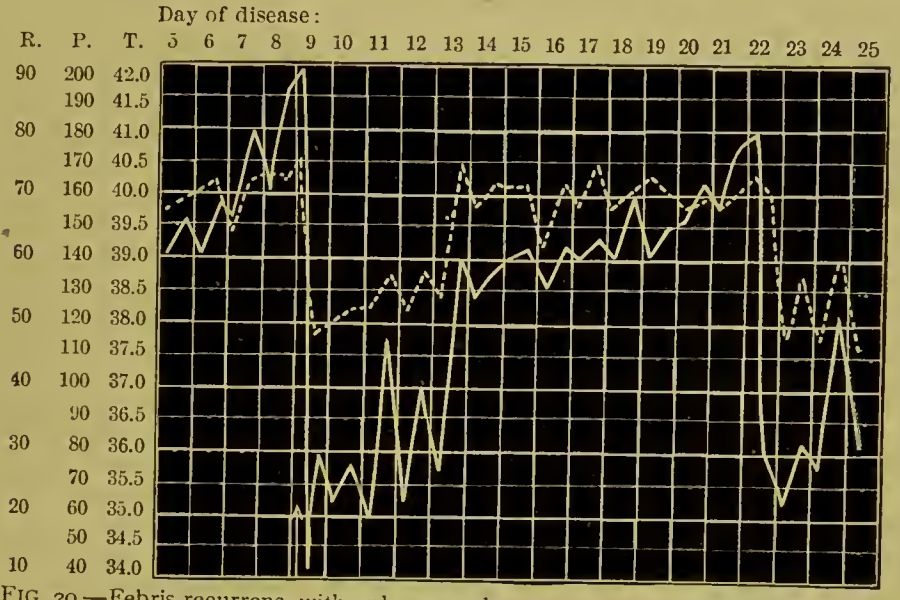


FIG. 20.—Febris recurrens, with only one relapse. Arrow at a collapse-like crisis. Male, age 44.

ture of that side is somewhat higher for a short time; then the temperature usually falls. Rare cases of hysteria exhibit a one-sided elevation of temperature with redness of the skin and perspiration.

2. Lowering of the Temperature.—This is the expression of local disturbance of the circulation. In heart-failure, also in collapse and near-approaching death, the extremities and also the nose become cool. Coolness of the affected limb is observed in venous thrombosis, in paralysis of long standing in consequence of diminished venous blood-current, and in arterial embolism and thrombosis.

PART III.
SPECIAL DIAGNOSIS.

CHAPTER IV.

EXAMINATION OF THE RESPIRATORY APPARATUS.

EXAMINATION OF THE NOSE AND LARYNX.

1. The Nose.—In making a local examination of the nose we employ *Inspection* and sometimes also *Palpation*. The inspection is external and internal: we look for asymmetry and other deformities and defects, and then at the shape of the nasal entrance [nostril]; and also we note the quality of the secretions.

Symptomatically, important anomalies of the nose are: uniformly swollen nose; the thickening, however, is most marked at the entrance (scrofulosis);[1] saddle-nose, caused by syphilitic periostitis, with exfoliation of pieces of bone. The syphilitic coryza (nasal catarrh) of the newly-born is associated with a peculiar snuffling sound.[1]

As regards the internal inspection, without the aid of instruments we can only examine the entrance into the nose. This only rarely shows characteristic alterations. For the inspection of the deeper parts a reflector and a nasal speculum are necessary. (See the paragraph in the Appendix upon Rhinoscopy.)

To the semiology of the affections of the nose belong the following symptoms: *fœtor ex ore* (ozena, ulcerations); occlusion, with respiration through the mouth, with obstruction of the nose or in the nasal cavity; speaking through the nose occurs under the same conditions, but also when there is paralysis of the soft palate. This also occurs when there is an abnormal communication between the mouth and nose (cleft palate). Dilatation and motion of the wings of the nose occurs in dyspnea.[2]

Lastly, there is nose-bleeding [epistaxis], which is usually without any significance. But it may be caused by some severe local or general affection (tumors, aneurysm, deep ulcers, hemophilia, temporary hemorrhagic disease). Nose-bleeding may be overlooked if it occur in deep sleep or in stupor (in acute infectious diseases), the blood flowing backward into the pharynx, through the esophagus, into the stomach. In this case there may be hematemesis, which may lead to an error in diagnosis.

Acute muco-purulent and purulent catarrh of the nose is symptomatic in measles, diphtheria, and equinia. Chronic catarrh is a

[1] See below.

[2] See below.

common symptom of scrofula (in which disease the whole nose is often swollen) and of syphilis. In the former disease there is sometimes an inflammatory thickening of the whole nose, particularly of its lower walls. Inflammation of an acute form, with very foul-smelling and ill-looking secretion, most frequently indicates diphtheria of the nose and pharynx. If it is chronic it may be due to catarrhal or specific ulcers.

Among the conditions which particularly demand an examination of the nose, we mention acquired and hereditary syphilis, bronchial asthma, supraorbital neuralgia, hemicrania, certain affections of the eyes and ears.

Regarding the further details upon the subjects of this paragraph we refer to the respective special works. As regards palpation of the posterior nares. see works upon Surgery.

Palpation of the interior of the nose may be necessary (see works upon Surgery).

2. The Larynx.—The larynx is examined with reference to its functions (voice, cough, breathing) and the local appearances; the latter include the external and internal examination.[1]

(*a*) **Function.**—The *voice* is changed in all affections of the larynx. It may be muffled, rough, hoarse, even to the entire loss of voice—"aphonia." In severe diseases it may have a whistling or sibilant (strident) quality: this indicates stenosis of the larynx; or it is very hoarse and deep: this points to deep-seated ulceration.

In diseases of the larynx the *cough* is hoarse, loud, or barking. In extensive destruction and in certain paralyses of the crico-arytenoid muscles cough is either more difficult or is impossible, since the power to close the glottis preceding the cough, as is normally the case, is wanting.[2]

Breathing is obstructed in all conditions that narrow the larynx, as in inflammation resulting in hypertrophy, in new formations, in scars with contraction. Then there is an inspiratory and expiratory dyspnea,[3] and a peculiar noise of stenosis, "stridor laryngeus." In marked stenosis, especially when the thorax is flexible, as in children, there is a drawing in of the lower part of the thorax in front in the region of the insertion of the diaphragm.[4]

Stenosis only in inspiration, causing inspiratory dyspnea, is observed in paralysis of the crico-arytenoid muscles, the dilators of the larynx.

Laryngeal stenosis is distinguished from *tracheal stenosis* at the first glance in that in the former condition the larynx moves up and down simultaneously with each inspiration and expiration, and the neck is stretched to the fullest extent, while in the latter the larynx remains quiet and the head is often somewhat bent forward.

(*b*) **Local Examination.**—**The external examination** is made with reference to pain, to deformities revealed to the sight or touch (these are very rare, resulting from destruction by periostitis), and laryngeal fremitus.

Laryngeal fremitus is a trembling of the thyroid cartilage during

[1] See also under Sputum.
[2] See Cough.
[3] See Dyspnea.
[4] See Anomalies of Respiration.

speech. It is stronger or weaker on one side in unilateral paralysis. It has no special diagnostic value.

The Internal Examination.—By great care, and in the case of patients who have themselves under good control, sometimes the entrance to the larynx and the tissues even as far as the glottis can be *touched.* This method, however, has now little value, since it has been entirely superseded by the examination with the laryngeal mirror, which is the best means of examining the larynx.[1]

In inflammatory conditions patients complain of *pain in speaking*, but sometimes, even with severe disturbances, there is no pain; now and then there is dyspnea, especially on exertion. *Pain in swallowing* in chronic diseases of the larynx frequently indicates serious conditions—extension of new formation (carcinoma, tuberculosis) toward the esophagus or destructive suppuration.

The leading *symptomatic indications of diseases of the larynx* with reference to other possible internal diseases are as follows: Acute laryngitis, with manifestations of an acute infectious disease, points especially to measles, croup, and also to small-pox; in chronic laryngitis, tuberculosis, syphilis, or a purely local disease of the larynx may be present; constriction by scars suggests, in the first place, syphilis, and also lupus. Of *paralyses*, paralysis of the recurrent nerve is of special diagnostic importance, since it often arises from *pressure upon nerves*, especially upon the left side from aneurysm of the aorta, carcinoma of the esophagus, tumors of all kinds in the mediastinum. Certain paralyses indicate hysteria.[2]

EXAMINATION OF THE LUNGS.

Topographical Anatomy of the Thorax.

For localizing the surface of the chest with reference to height and depth we make use partly of anatomical prominences and partly (for determining the breadth) of certain local lines which we think of as drawn upon the surface of the thorax.

Upon the front side of the thorax are the important anatomical regions: the fossa supraclavicularis (above the clavicle and bounded by the sterno-cleido-mastoid and trapezius muscles) and the fossa infraclavicularis. The latter has no distinct lower boundary. We understand it as the region immediately below the clavicle, about as far as to the second rib. From the second rib downward we designate the height by the ribs and intercostal spaces, as above the fourth, under the fourth rib, the fourth intercostal space. The number of the particular rib is determined by counting from the second rib downward. It is always easy to find this rib: it is in articulation with the sternum exactly where the manubrium and corpus sterni unite, ordinarily forming a very slight angle (angulus Ludovici), and this place is plainly to be felt, and often seen, as a cross-line or prominence. We feel for this prominence and find the second rib to be its prolongation. We count the ribs from that downward, feeling obliquely outward as we go down. Morenheim's depression [the outer part of the infraclavicular depres-

[1] Regarding its use, see the Appendix.

[2] See below.

sion] and the so-called Sibson's furrow (the under border of the pectoralis major) are sometimes, although not very practically, useful as points for locating internal organs.

For determining the breadth the vertical lines now to be mentioned are useful (the subject is supposed to be standing): the middle line, drawn through the sternum; the two sternal lines, drawn parallel along the sides of the sternum; the mammillary lines, drawn through the male nipple; and the parasternal lines, drawn midway between the sternal and the mammillary lines.

On the two sides we determine the height by the ribs, which we count in front, and the breadth by the middle axillary line (drawn through the middle of the axilla, the arm being extended sidewise), the anterior and posterior axillary lines (drawn perpendicularly from the points where the pectoralis major and latissimus dorsi muscles leave the thorax, with the arm raised sidewise to the horizontal).

Upon the back we name the fossa supraspinata; above that, the suprascapular space, the fossa infraspinata, the interscapular space, between the two scapulæ, the infrascapular space, under the shoulder-blades. Exact determination of height is made by counting the ribs, which, however, are difficult to count, especially in fat persons. They can be determined by three methods:

(*a*) By counting the vertebral prominences from the vertebra prominens (the seventh cervical).

(*b*) By counting from the lower angle of the scapula: this overhangs the seventh rib in the average person when the shoulders hang comfortably and the arms rest against the chest with the forearms folded lightly.

(*c*) By the point of the twelfth rib, which is easily felt (the best way for the lower ribs).

Moreover, we have the scapular line, which is drawn upon the two sides of the spine through the lower angle of the scapulæ (at the point already mentioned under (*b*)).

It is to be observed that some of the vertical lines are not determined exactly. This is true regarding the mammillary line (always very important) more than any other. In women it is generally very variable. On this account it is always to be thought of as drawn upon a male thorax. But even in the male the nipple is an uncertain point. By much practice the eye is cultivated so as to recognize what is to be regarded as the average location of the nipple in the male, and by this we must always correct the mammillary line. The various attempts to substitute other lines for this one have failed.

The designation "infrascapular space" is little used. The expressions "right, left, behind, below," correspond to it, and are much to be recommended: behind or below the right, the left, scapula.

The Anatomical Boundaries of the Lungs with Reference to the Thorax.

In front the lungs reach to the sixth, and behind to the tenth, rib, and are almost everywhere directly in contact with the chest-wall.

They are not in contact with the chest-wall in the neighborhood of the heart nor behind a small portion of the upper part of the sternum.

The accompanying figure exhibits the anatomical boundaries of the lungs. They project with their summits into the fossa clavicularis from 3 to 5 cm. above the clavicle, and with their inner anterior borders converging downward, so that behind the angulus Ludovici—not exactly behind the middle of the sternum, but a little to the left—they come to lie very closely to each other; then they continue parallel downward to the insertion of the fourth rib. From there the inner border of the right lung proceeds still farther downward to the top of the insertion of the fifth rib, and then gradually bends toward the right,

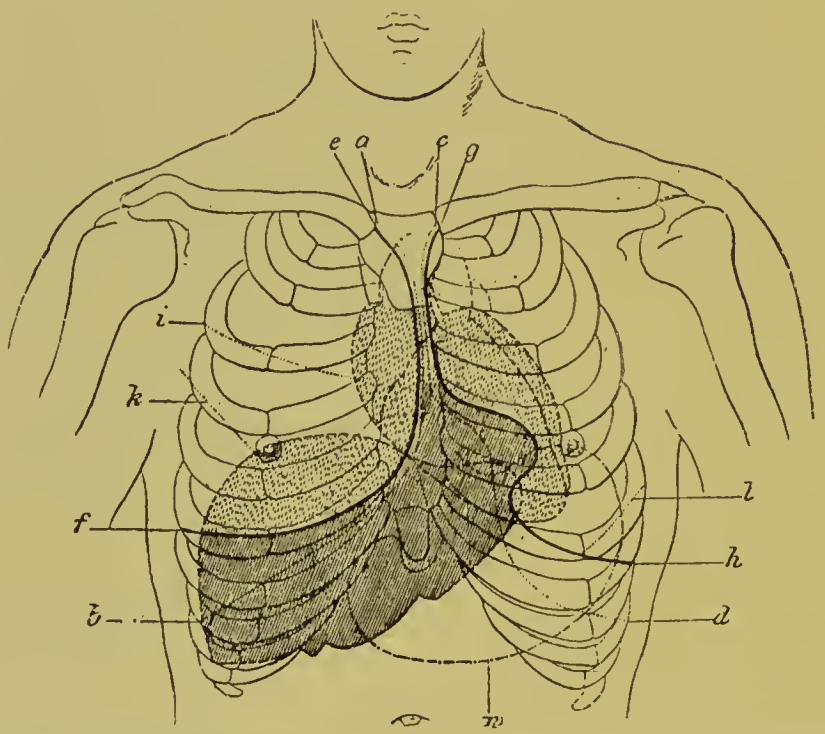


FIG. 21.—Position of the thoracic viscera, of the stomach, and of the liver, from in front. The portions of the heart and liver which are drawn with unbroken hatched lines represent the parietal portions of those organs. The portions that are not in contact with the chest-wall, but are covered by the lungs, are represented by broken (clear) hatched lines.

The line *e f*, border of the right lung; *g h*, border of the left lung; dotted lines (. . . .) *a b* and *c d*, the boundaries of the complementary pleural space; *i*, the boundary between the right upper and middle lobes; *k*, the boundary between the right middle and lower lobes of the lung; *l*, boundary between the left upper and lower lobes; *w*, greater curvature of the stomach (Weil-Luschka).

so that it follows along the sixth rib, on the upper border of which it meets the mammillary line. Then it continues approaching the horizontal (in the upright posture), so that in the middle axillary line it lies upon the seventh or eighth rib, in the scapular line upon the tenth rib (this location on the dead body is about 1 cm. higher than in quiet respiration in the living subject). On the left side the border of the lung bends sharply round from the fourth rib to give place to the heart, continues behind the fourth rib as far as the left parasternal line, then bends vertically downward, making a small bow which converges toward the right; then, sharply bending again behind the sixth rib, so

as to pass the mammillary line under the sixth rib (hence somewhat lower than on the right side), it passes the axillary line between the seventh and eighth, and the scapular line at the tenth, rib.

The boundaries of the lungs are different according to age, as well as in individuals. (See section on Percussion of the Lungs.)

The boundaries of the pleural sacs—that is, the lines on which the pleura costalis (sternalis) leaves the wall of the thorax and bends inward—agree in reality with the course of the inner borders of the lung. But along the lower borders of the lungs and at the cardiac concavity the pleural space extends considerably beyond the border of the lungs (in quiet breathing), making the sinus phrenico-costalis and the com-

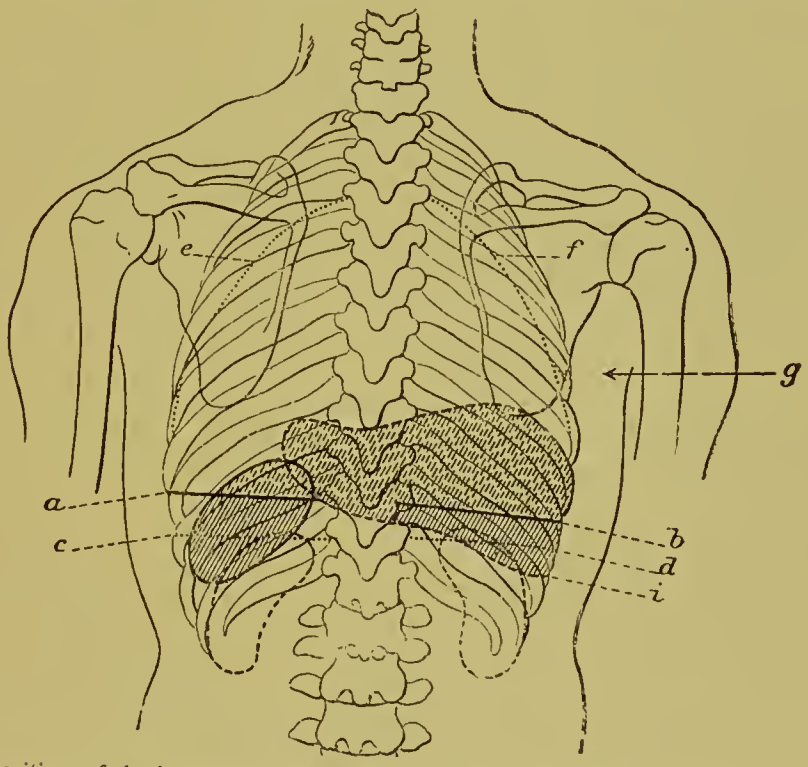


FIG. 22.—Position of the lungs, liver, spleen, and kidneys seen from behind. The liver and spleen are represented by the same hatching as in Fig. 21.

a b, the lower border of the lungs; *c d* (. . . .), complementary space; *i* (dotted line) (broken line), border of the liver; *e f* (dotted line), boundary between the upper and lower lobes of the lungs; *g*, boundary between the upper and middle lobes of the right lung (Weil-Luschka).

plementary pleural sinus. The size of these corresponds with the form. The largest is the complementary pleural sinus in the two axillary lines. This is there about 10 cm. high.

The pleural sinuses are therefore important, since into them extend the lungs at every deep inspiration, and also in the pathological, chronic inflation, *emphysema pulmonum;* and also—because in them fluid effusions into the pleural cavity ordinarily first accumulate.

The under surface of the lungs rests directly upon the diaphragm. The diaphragm in the dead body rises at its highest part, as a dome, about as high as the insertion of the fourth rib, a little higher upon the right than upon the left side. The average situation of the dome of the diaphragm in life, during quiet breathing, is a little lower.

Finally, it is necessary to mention the course of the boundaries of the lobes of the lungs, since they sometimes have an important part in diagnosis: At the back, near the spine, the boundary between the upper and lower lobes is at the height of the lower angle of the scapula; upon the left it gradually slopes forward and outward in such a way that in the axillary line it stands at the fourth rib, and meets the lower border of the lung (that is, at the sixth rib) in the mammillary line. On the right side the boundary-line divides near the outer border of the scapula into two diverging lines—the line between the upper and middle lobes and that between the middle and lower lobes. The former proceeds at first behind the third rib, and terminates at the inner border of the lung at the insertion of the fourth rib; the latter meets the lower border of the lung somewhat within the mammillary line, and therefore behind the sixth rib.

Hence, in front upon the right side we have the upper lobe about at the third intercostal space; from there downward, really the middle lobe; in front on the left side, for the whole distance, we really have the upper lobe; on the right side we have the middle lobe above and the lower lobe below; on the left side we have the lower lobe; behind we have only the apices, formed by the upper lobes; all the rest is lower lobe.

Inspection of the Thorax.

The examination of the thoracic organs must always begin with the inspection of the thorax. Nothing is more faulty than to take up some other method of examination first. Inspection of the thorax is important because a very large number of the diseases of the lungs and pleura manifest themselves in the form of the chest-cavity and a change of the respiration. Certain diseases of the internal organs have a causal relation to changes in the form of the thorax. In other cases, as it appears, a given form of thorax accompanies a "disposition" of the lungs to certain diseases (emphysema, phthisis). It is very probable, although it is difficult positively to establish, that sometimes the thorax by its form either causes or favors the development of the given disease. Moreover, we know that there are deformities of the chest which in other ways injure or render useless the thoracic organs; there are such also as have no influence upon the lungs or heart.

Method of Procedure.—During inspection (as in all examinations of the thorax) attention must be given to having the patient straight, but without undue muscular tension. The light should fall symmetrically upon the front or back, whichever is under examination; the eyes of the examiner should, if possible, be directly before the middle line of the body. The general structure of the thorax (and neck) should first be considered, next possible peculiarities, then the motions of respiration, first during quiet, then deeper, respiration.

1. **Normal Form of Thorax and Normal Respiration.**—In a well-constructed thorax we expect, first, perfect symmetry. However, this is departed from almost always normally, in that there is a very slight curvature of the dorsal vertebræ toward the right. Moreover, the clavicular depressions may be only slightly indicated; the angulus

Ludovici [also called the angle of Louis] (the angle formed by the junction of the manubrium and corpus sterni) may just be recognizable; the true ribs should so leave the sternum that from the top downward there is increasing obliquity, making the angle formed by the two opposite bendings of the ribs, "the epigastric angle," almost a right angle. The thorax should be well developed; the scapulæ in the upright position should lie flat upon it; the intercostal spaces should be visible only at the lower ribs; finally, the dimensions of the chest and the size of the body should have a certain relation to each other. Very seldom does the normal thorax correspond to this ideal, and there are many departures from it in persons who are perfectly sound. Some "physiological" departures may be mentioned: a slight asymmetry in a gradually-acquired spinal curvature or a deformity of the ribs, self-established; further, a peculiar form of thorax, where the upper part is somewhat shallow, but the lower of increasing depth, so that the lower aperture of the thorax is very large; also more marked angle of Louis (Braune); again, in a shorter thorax, a more obtuse epigastric angle may sometimes be observed in healthy persons (hence also without signs of emphysema).[1] The supraclavicular depressions are often both deepened, with the apices of the lungs entirely normal (unequal deepening of them is, however, very suspicious of tuberculosis);[2] single ribs, more frequently the second, third, also the fourth, on account of greater curvature sometimes project more in front; on the other hand, the lower ribs will often be found pressed into the side and from there flattened forward; and other variations. The boundary between the unsymmetrical and the pathological form of chest is much confused; it can only be recognized in the individual case by attention to the location and function of the thoracic organs.

Normal breathing takes place in this wise: inspiration only is active —that is, is accomplished by muscular action; expiration, on the contrary, is produced wholly by the elasticity of the lungs, the elasticity and the weight of the chest-wall, and the pressure of the abdominal organs upon the diaphragm. The number of respirations to the minute in the new-born is about 44; at five years, about 26; from the twentieth year, about 16 to 20. It is very easily influenced by a number of conditions: in sitting and standing it is somewhat higher than in lying; it is increased by bodily activity and psychical impressions. Therefore it can only be determined during perfect quiet, with the attention withdrawn from the examiner, or during sleep. For counting it, it is generally most advantageous to lay the hand lightly upon the chest (or upon the epigastrium).

The breathing is generally regular, and the single breaths are of equal strength; but under the influence of the slightest psychical disturbance they easily become irregular and unequal. Many persons of sound health, as snorers in sleep, often breathe irregularly or unequally deep. Breathing is either exactly or very nearly symmetrical, though the left side frequently inclines to breathe a trifle stronger.

The *inspiratory enlargement of the thorax* is occasioned by the elevation of the ribs and the sternum and the simultaneous drawing of the former upward and outward (intercostales externi and interni muscles

[1] See below.

[2] See below.

—"*costal breathing*"); moreover, by the contraction of the diaphragm, and hence flattening of its dome. The latter movement at the same time draws down the intestines, and so with every inspiration the whole anterior wall of the abdomen projects, but especially the epigastrium ("diaphragmatic," or abdominal, breathing). The combination of costal and diaphragmatic breathing varies in the two sexes: in the male the latter, and in the female the former, preponderates. But in aged females with firm thoracic walls diaphragmatic breathing increases; while, on the other hand, male as well as female children incline to the costal type of breathing. From this it seems that the degree of flexibility of the thorax influences the kind of breathing.

In the costal breathing of women, even in quiet respiration, the scaleni muscles (elevators of the first and second ribs) take a part, while in men these muscles belong to the auxiliary muscles of respiration.[1]

Diaphragmatic Phenomenon (Litten).—This peculiar and very striking phenomenon can only be observed by keeping rather closely to the proceeding which the discoverer has indicated:

The person to be examined should be undressed to the middle of the abdomen, and then should lie down as nearly as possible in a horizontal position, with the feet toward the light. The room should be lighted only from one side. In the daytime, therefore, one should select a room with only one window, or, if there be more than one, all but one must be darkened. The patient is asked to breathe deeply, and the breathing must be diaphragmatic. The attention of the observer is directed to that part of the thorax which is below the fifth rib. The observer stands about one and a half to three steps from the side of the person examined or from a position midway between the side and feet. In most people, but not at all in stout persons, a shadow-like line ascends and descends with each respiration. This line is only present in the intercostal spaces, but as it crosses the ribs diagonally it appears in several intercostal spaces at the same time, and moves regularly up and down, and, in spite of the interruption by the ribs, it gives the impression of a continuous line. It is seen most distinctly near the anterior axillary line between the seventh and ninth ribs. In some people it can be followed from there far to the front and even round to the back. From the illumination necessary to observe this phenomenon we conclude that the skin slopes toward the head. This sloping must of course be connected with diaphragmatic respiration or with the displacement of the edge of the lungs, the more so as it always coincides exactly with the boundary of the lungs as made out by percussion. The falling off of the surface of the skin taking place from below upward, as we have mentioned above, it is therefore probable that it corresponds, as Litten thinks, with the separation of the diaphragm from the wall of the thorax. It is impossible to see in this phenomenon that portion of the edge of the lungs which goes down into the complementary space, for this would produce a slope from above downward.

The value of the phenomenon for the diagnosis of the extent of

[1] See Auxiliary Respiratory Muscles.

diaphragmatic respiration is, in my opinion, impaired by the fact that the phenomenon can be observed even in healthy people, frequently in only a small part—namely, that which is directed from above downward. There are not many cases in which it is seen moving up and down for six or seven centimeters, as is stated by Litten. If on one side the diaphragm does not move, and if the diaphragm is forced downward, the phenomenon will not be seen on that side, and consequently it is not observed in paralysis of the diaphragm and when there is considerable exudation and transudation of the pleura in pneumothorax, in pneumonia of the lower lobes, and also sometimes in subphrenic peritonitis. But it is well known that the last-named disease sometimes does not interfere with the contractions of the diaphragm, and in such a case the existence of the diaphragmatic phenomenon would decide the differential diagnosis against exudation in the pleura. But I have never yet seen such a case.

2. Pathological Forms of Thorax.—(*a*) **The Inflated or Emphysematous Thorax.**—This refers to a chronic symmetrical expansion in all directions, conforming somewhat to the form of the chest during inspiration (the inspiratory position). The antero-posterior (the sterno-vertebral) diameter is increased. In many cases it appears as if the thorax became enlarged, especially at about the height of the middle of the sternum, making a barrel-shaped chest; however, this may be entirely wanting. The ribs are generally strong, and are at right angles to the sternum, hence the epigastric angle is larger than normal; the thorax is generally short. Frequently the angle of Louis is very prominent.

The supraclavicular depressions may vary very much: sometimes they are deepened; again, shallow or even projecting like pillows (the latter condition obtaining in emphysema of the upper part of the lungs). The lower intercostal spaces are sometimes drawn in during inspiration (inspiratory drawing-in).[1]

In the emphysematous thorax the breathing is so changed that the expiration is both slower and imperfect in consequence of the diminished elasticity of the lungs; it is prolonged, and in marked emphysema it is assisted by muscular action, especially by the transversalis abdominis and the quadratus lumborum. We can then plainly see the abdominal wall energetically flattened, and we are directly impressed with the idea that the thorax is forcibly expanded. But the inspiration is also altered in consequence of the rigidity of the chest-wall; ordinary costal breathing is wanting; it is very imperfect; and in its place we notice that the front of the chest, as a whole, has been drawn up by the powerful action of the sterno-cleido-mastoidei muscles. Consequently, in emphysema we have the breathing rendered difficult; in severe cases it may become so to a high degree.[2]

The typical emphysematous thorax points almost with certainty to emphysema, and hence its name; however, we must guard against the mistake of calling every short chest an emphysematous one. On the contrary, also, we not infrequently find a general emphysema of the lungs in a chest that has no trace of the "emphysematous" form. Active expiration, expiratory dyspnea, is much more characteristic

[1] See below, p. 87.

[2] See Dyspnea.

than the form of the thorax; besides emphysema, it exists in no other condition except certain diseases of the larynx.[1]

(*b*) **The Paralytic or Phthisical Thorax.**—This is the direct opposite of the preceding: it is flat, especially in the upper part; is often also narrow; the intercostal spaces are wide; the ribs are generally delicate, are sharply inclined downward from the sternum, and hence must be bent at a sharp angle again in order to come back to the vertebræ. This sloping from the sternum makes the epigastric angle very sharp; the chest, as a whole, chiefly in consequence of the course of the ribs, is long. The angle of Louis is often very marked. The depressions are generally deep. The shoulder-blades frequently stand out like wings.

Quiet breathing may be almost normal, but on exertion it is generally immediately very much increased in frequency; it is shallow; even in women the costal type is often wanting, especially at the upper part of the chest.

This form of chest corresponds with that of tuberculosis. A well-marked paralytic thorax, except where phthisis of the lungs has early developed, is very infrequently seen; but yet this disease occurs very often where the phthisical thorax is wholly absent—indeed, with an emphysematous thorax. In a paralytic thorax, with phthisis already developed, by means of the latter the form of the thorax and the breathing will become essentially and variously changed.[2]

But one must be very careful not to conclude that a thorax narrow from great emaciation, and especially one that appears flat, is a paralytic one. For example, a beginner is apt to find that a patient convalescent from typhoid fever has a paralytic chest. Strictly speaking also, every plain or flattened thorax is not to be called a paralytic one. Moreover, emaciation and flattening of the upper parts of the chest in cases of developed phthisis frequently render the thorax paralytic, which it originally was not.

(*c*) **One-sided expansion of the thorax,** a relatively infrequent affection, occurs in disease or functional loss of the opposite lung. The dilated side is then the seat of the so-called "vicarious emphysema" of the lung. This is distinguished from true emphysema by the absence of expiratory dyspnea.

The dilated side is much more frequently the diseased one. The widening of the chest-cavity is more plainly seen from the front than from behind. Very frequently the mamma and the scapula are further removed from the median line than upon the normal side. The intercostal spaces are level or are projecting; in contrast with this, the diseased side drags after the other—that is, in inspiration it rises later and less than the sound side, and it may even not rise at all. Hence the spinal column is sometimes bent toward the diseased side.

Marked expansion is met with in pneumothorax and in extensive pleuritic exudation, while the development of the latter usually first manifests itself by expansion and lagging behind at the posterior and lower part of the chest. A very slight expansion of one-half of the chest is, moreover, sometimes seen in croupous pneumonia of the whole of the affected lung.

[1] See Dyspnea.

[2] See above under (*a*) and below under (*d*).

Circumscribed forward expansion of the chest occurs especially with tumors of the pleura, and is sometimes humped, and again uniform; empyema which inclines to breaking through pushes the affected region prominently forward, and at the same time the skin is generally edematous. Encapsulated pleuritic exudations or circumscribed pneumothorax seldom causes expansion, yet the first cause a smoothing out of the neighboring intercostal spaces, besides lagging behind. Local projections, moreover, sometimes occur from inflammatory affections or neoplasms of the ribs or the subcutaneous cellular tissue.

Local expansions of the thorax are seen in cases of enlargement of other organs. The cardiac region may be bulged out in enlargement of the heart or distention of the pericardium;[1] a marked enlargement of the liver may press out the lower ribs on the right side, and enlargement of the spleen on the left; and sometimes, especially in children, a very marked expansion of the whole lower part of the thorax, an enlargement of the lower aperture of the chest, is observed in cases of considerable expansion of the whole or the upper part of the abdomen (meteorismus, ascites, peritonitis, tumors). Then the upper part of the chest seems quite small in comparison with the lower part; the whole trunk is hence shaped like a bee. From the drawing up of the diaphragm there results interference with diaphragmatic breathing, and generally there is severe dyspnea.

The extent to which the thoracic wall is driven forward, if caused by pleuritic exudation, depends to a large extent upon the degree of flexibility of the thorax. If the wall is soft, as is the case with children, the expansion is very pronounced; if rigid, as in subjects of emphysema, sometimes a very large pleuritic exudate causes no noticeable expansion. Therefore, while we expect in general that an extensive pleuritic exudate will manifest itself by an enlargement of the affected side of the chest, yet where the walls are rigid we must not conclude from the absence of expansion that there is no exudate.

(*d*) **Drawing-in or Shrinking of One Side.**—This is seen more or less frequently as a symmetrical drawing-in of the whole side, so that the affected side is altogether smaller than the other; the ribs are close together, and in the lower part they may even overlap like shingles on a roof. The shoulder of that side hangs down; the mamma and scapula are nearer the median line. The *spinal column* is curved with its convexity toward the healthy side; hence the whole carriage is affected. There is diminished breathing or no breathing at all on the side drawn in; on the healthy side there develops a vicarious emphysema. This condition is observed in recovery from extensive pleuritic exudations and in long-continued contraction of the lungs.

In pleurisy it is the loss of elasticity and thickening of the pleura, with adhesions of pleural surfaces, in shrinking of the lungs, and the development of connective tissue in the lungs, which not alone hinder the lungs from following the inspiratory expansion of the thorax, but from the tendency to contract, as in scars of the skin, draws in the chest-wall. This inward traction, however, does not concern the thorax alone: the mediastinum, heart, and diaphragm are pulled

[1] See under Examination of the Heart.

toward the sunken side. Hence there is displacement of the heart toward the diseased side and the diaphragm is high in the chest.

More frequently there is an unequal degree or a partial shrinking on the affected side; with it also is always connected a more or less marked lagging. It is most frequently observed above in front, here sometimes noticeable at the first commencement as a deepening of the supraclavicular depression (an important symptom of contraction of the apex of the lung from tuberculosis). Again, a partial drawing-in is often seen, most frequently low down posteriorly, after the disappearance of a small pleuritic exudate. But there may be shrinking of any part of the chest-wall, as after gangrene or abscesses of the lungs.

One must be careful not to confound a deformity of the chest from disease of the thoracic organs with deformities that are dependent on a primary bending of the spine and thorax. Concerning these see the following section.

A repaired fracture of the ribs may also cause deformity; a fracture of the clavicle which has healed with an angle forward may deepen the supra- and infraclavicular depressions, and so deceive one; one-sided defect or atrophy of the pectoralis major of course flattens that side. All of these cases may be excluded by a more careful examination.

(*c*) **Alterations of the Form of the Chest by Primary Deformity of the Skeleton.**—*Kyphosis*, or bending backward, and *scoliosis*, the bending sidewise, of the spine, but, still more, the combination of both, *kyphoscoliosis*, sometimes occasion deformities of the chest that are enormous. Most frequently one side is smaller in front, while the other side appears to be enlarged; and the picture of one-sided contraction of pleura or lung is more complete from the dragging-after of the smaller side. In consequence of a peculiar twist of the spine and its effect upon the course of the ribs the back is generally very crooked. This is spoken of more particularly in works upon surgery. The organs of the chest are almost always displaced from their normal position. The lungs are very much impaired in their function. Such patients become short-breathed on the slightest exertion. In diseases of the thoracic organs, and also in acute infectious diseases, these patients are exposed to greater risk than others. Whether in such cases we have to deal really with a primary deformity of the chest or with a contraction of the lung or pleura is generally made clear by the examination of the spine. Sometimes, however, a very careful examination of the skeleton and of the thoracic organs is necessary to answer this question; and in some cases of long-existing deformity even this differential diagnosis may be impossible. The distinction of the different kinds of spinal curvature and their origin belongs to surgery.

Rachitis is frequently the cause of such deformities, but it may also cause all other possible bendings of the chest. Of these, especially characteristic are—1. The *rachitic chest*, a thickening of the point of transition from the cartilage to the bony ribs. The several prominences arising from it form on both sides of the sternum a line passing as an arch outward and downward. 2. *The pigeon-chest.* The chest seems to be compressed sidewise and pressed forward. The ribs run sharply

backward from the front, so that the sternum stands forward like the keel of a ship, the sternovertebral measurement being much increased. 3. *A circular drawing-in in the neighborhood of the costal attachment of the diaphragm and above it.* This retraction is in part directly produced by the diaphragm, because the softened ribs do not offer sufficient resistance to its contractions. The retraction, however, partly results from the circumstance that the thorax sinks in just above the point of insertion of the diaphragm in consequence of the inspiratory lowering of the internal pressure. If dyspnea exists, and consequently increased action of the diaphragm, the retractions are increased.

Funnel-breast (Fig. 23).—This deformity consists in a sinking-in of

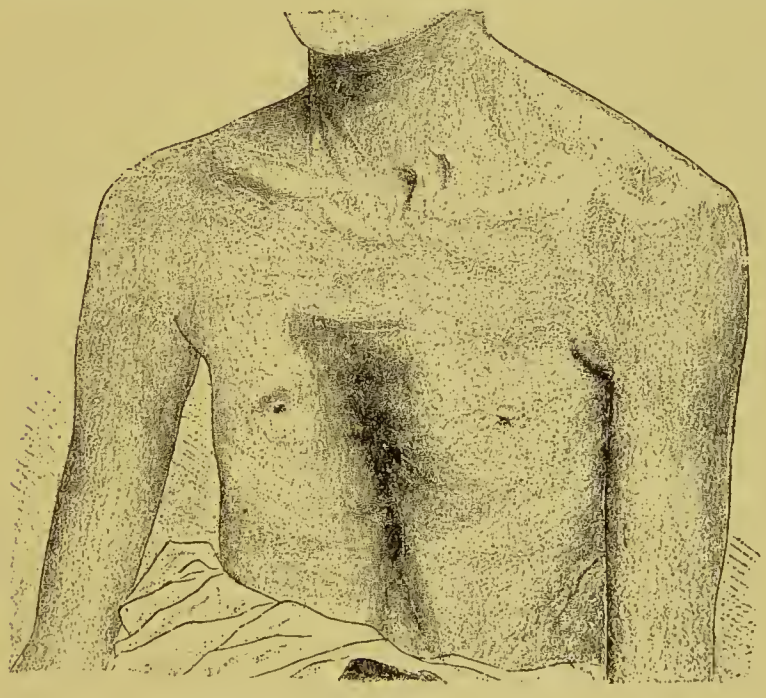


FIG. 23.—Funnel-breast (Ebstein).

the sternum, especially of the lower portion of it; it may be very considerable (as much as 7 cm.). The affection is generally congenital, and, according to our experience, in very marked cases it may prove a hindrance to respiration. *Shoemakers' breast* exhibits a sort of acquired funnel-breast, caused by pressure of tools against the lower part of the sternum and the xiphoid cartilage; the depression never becomes very great, and involves only the cartilage; it has no pathological significance.

According to recent experience, the funnel-breast sometimes is observed in several branches of a family. In individual cases it occurs as a sign of degeneration with other errors of development, or associated with neuropathic or psychopathic disease or hereditary taint.

3. Anomalies of Respiration.—In the preceding section the anomalies of breathing which accompany the several pathological forms of thorax have been briefly referred to. But these require a further separate description. In giving this it will not be possible to avoid a partial repetition of what has already been said.

(*a*) **Anomalies of the Manner of Breathing.**—The type of breathing which, as has been mentioned above, in the normal human being is typically different in the two sexes, and is denominated costal and costo-abdominal, may be influenced by a number of different pathological conditions:

1. The activity of the diaphragm, from some cause or other, may be restricted or entirely stopped; it may then be replaced by increased thoracic breathing; this causes the costal type peculiar to women to be still more prominent, while the male type is reversed; instead of the abdominal predominating, the costal becomes predominant or entirely prevails—that is, may take on the female type.

Such a restriction or prevention of the action of the diaphragm is occasioned by pain or mechanical restraint, or by weakness or paralysis of the diaphragm. Such is the action of all inflammations of the abdominal or pleural cavities in case they involve the corresponding serous covering of the diaphragm, markedly impairing diaphragmatic breathing. They often act so because they are painful; but also sometimes, especially in inflammation of the diaphragmatic peritoneum, actual paralysis of the diaphragm quickly develops, which is recognized by the entire disappearance of abdominal breathing.[1] This takes place quite commonly in diffuse peritonitis; it is, however, also sometimes the only symptom of a beginning local "subphrenic" peritonitis. Marked distention of the abdomen by tumors, fluid, and accumulations of gas in the intestines hinders diaphragmatic breathing in a high degree. Finally, there occurs paralysis of the diaphragm in organic diseases of the nervous system (bulbar paralysis, neuritis of the phrenic nerve in the various forms of multiple neuritis), as well as a manifestation of functional neurosis (hysteria).

The action of the diaphragm is recognized, as has frequently been mentioned, by the protrusion of the epigastrium during inspiration. Of course this does not take place when there is no contraction. In complete paralysis the diaphragm is sometimes even completely sucked into the thorax; in hysteria, during inspiration, the epigastrium sometimes sinks in extraordinarily deep. One-sided failure of action of the diaphragm may also occasionally be made out.[2]

2. But sometimes, also, hindered thoracic breathing may be replaced by increased diaphragmatic breathing; hence in such a case, if the patient is a female, the type of breathing is changed—that is, abdominal breathing predominates instead of costal.

Therefore, in very rigid thorax (emphysema), sometimes also in women, diaphragmatic breathing predominates. Here belong paralysis of the muscles of inspiration (bulbar paralysis) and myositis ossificans (rare), since the latter causes a rigid condition of the thorax. A disease of the skin at present well known, but rare, scleroderma, may, if located upon the thorax, also entirely abolish thoracic breathing.

It has been shown above, under Emphysematous Thorax, how, in lieu of the peculiar costal breathing, this may in part be replaced by the movement of the thorax as a whole by the (auxiliary) muscles—the sterno-cleido-mastoidei.[3]

3. Asymmetry of breathing, which is occasioned as follows: the

[1] See p. 74. [2] See Palpation. [3] See below.

whole side or the upper or lower part of one side either (very rarely plainly) expands somewhat later than the opposite side or (most frequently) expands less strongly or not at all; which condition has already been mentioned several times.

Such a lagging may be caused by a unilateral painful affection of any kind; moreover, by all diseases of the thoracic organs which interfere with respiration upon one side. This "lagging behind" is a valuable symptom, especially in *phthisis* (lagging in the infraclavicular depression), also in the beginning of *pneumonia and pleurisy*, when other symptoms are wanting.[1]

(*b*) **Anomalies of Breathing as Regards Frequency and Rhythm.**—Diminished frequency of breathing may take place in all severe diseases of the brain and its meninges, hence in large hemorrhages, tumors, etc. and in all forms of meningitis; thereby exists always more or less dulness of intellect; the slowness of breathing may sometimes pass into the Cheyne-Stokes respiration.[2] Further, in acute infectious diseases with marked mental dulness the respiration may be slower; finally, it is generally so in the death-agony.

A very important form of diminished frequency of respiration is observed with stenosis of the upper air-passage; this belongs in the section on Dyspnea. Increased frequency of respiration as a pathological manifestation belongs, without exception, to a large group, which will also be discussed in the next section.

It has already been mentioned that we meet with temporary irregularity of breathing in healthy persons. It is of pathological, and generally of grave, import in all cases of marked mental dulness (as in apoplectic, uremic, and the coma of severe typhus), and very especially in the death-agony.

Forms of Periodic Respiration.—1. *Cheyne-Stokes Respiration.*—This is a kind of respiration in which, in pronounced cases, a group of respirations regularly alternates with a more or less prolonged pause of respiration, or *apnea*. The transition, however, from one state to the other is effected gradually, the period of respiration beginning and ending with shallow breathing. The patients, who in most cases are in a state of stupor, impress the observer by the circumstance that with regular pauses they make a few deep snorting or snoring, or perhaps sighing, respirations. If one observes a little closer, he sees that these deep respirations are followed by a few which become weaker and weaker, and then the respirations cease altogether. After a certain pause there is a short, scarcely perceptible respiration. This is followed immediately by a somewhat deeper one, and progressively the respirations become abnormal in depth, from which they slowly decline till a period of apnea is again reached. The pause in respiration may last a minute, and exceptionally even longer. [The translator recently observed a case following uremic convulsions in which the pause lasted ninety seconds.] The number of respirations in one period varies. Most frequently there are eight to twelve, which follow each other in about normal celerity, but at the beginning and end of the period of respiration they are sometimes somewhat slower. Occasion-

[1] See Palpation of the Thorax.
[2] See below.

ally there are, besides, some secondary symptoms: regular contraction of the pupils during apnea, and dilatation of them in the height of respiration; retardation of the pulse during apnea; isolated contraction of muscles at the end of apnea. Sahli has observed that patients become cyanotic at the beginning of respiration, and that the cyanosis increases till the height of the respiration is reached, which corresponds with the conduct of the pupils. Finally, we and also others have here and there observed a periodic change in the state of consciousness corresponding exactly with the respiration: patients already somewhat stupid become entirely unconscious during the period of apnea, and with the beginning and deepening of respiration they regularly revive, look around, and even speak.



FIG. 24.—Stethographic curve with Cheyne-Stokes breathing (after Reider).

Besides the perfect form of Cheyne-Stokes respiration, just described, there are also some less striking forms. It is not necessary that it comes to an audible deep breathing, or even to an abnormal depth of respiration; or the period of apnea may be very short. The apnea may also be missing, in such a manner that only deep and more superficial breathing alternates, in regular periods, with a uniform gradual transition. It seems to us that this also belongs to the Cheyne-Stokes form of respiration.

This phenomenon is by no means rare, particularly if we include the type of cases just described. It is principally observed in diseases of the brain, in severe disturbances of the circulation, and in toxical states. It occurs in meningitis with tumors of the brain, following cerebral hemorrhages, etc., and in severe weakness of the heart following diseases of that organ, particularly in fatty degeneration (Stokes); then fre-

quently in uremia, in cases of morphin-poisoning, in acute diffuse peritonitis, and, finally, in acute infectious diseases, particularly in typhoid fever. It is very seldom seen in persons suffering from slight forms of disease. There have been observed, however, traces of this anomaly of breathing during the sleep of healthy persons (Mosso).

We have, on the contrary, almost always to do with patients severely ill, often with stupefied or unconscious ones. Sometimes patients breathe in this way only during an otherwise normal sleep, and then the symptom seems to have proportionally less significance. Cheyne-Stokes respiration is frequently the precursor of the irregular, agonal respiration, and hence, from a diagnostic standpoint, it presages a bad turn. But still, that is by no means always the case, for, in the first place, it is sometimes observed for weeks and even months, especially during the sleep of persons suffering from heart and kidney disease. It also not seldom accompanies transitory toxical states, most frequently, in my experience, in uremia. The manner in which, and the circumstances under which, Cheyne-Stokes respiration occurs points to the supposition that its cause is in an alteration in the function of the respiratory center. Traube and others after him have attempted to find an explanation of the phenomenon. All these explanations result in the supposition of an alteration or diminution of the excitability of the respiratory center in the medulla oblongata, or in the supposition that the excitability of the oblongata becomes exhausted. But, in my opinion, no one has satisfactorily explained the peculiar periodicity of the respiration. When there certainly exists a diminution of excitability, and also a liability of the ganglia of the oblongata to become exhausted, as occurs shortly before death from any cause whatever, we simply observe that the respirations become less frequent and more superficial. But no one has yet succeeded in explaining the pauses, and particularly the successive increase of the depth of breathing, in the beginning of respiration after the pauses.

Biot's Respiration.—By this designation is understood periodic pauses in respiration, alternating with normal respirations more or less regular. The phenomenon, which is very rare, occurs most frequently in diseases of the brain, particularly in meningitis. The significance of the symptom is the same as that of Cheyne-Stokes' respiration. Beyond this one may doubt whether this is not a sub-species of Cheyne-Stokes respiration, or whether it is a phenomenon which is different in principle from it.

(*c*) **Difficult Breathing, Dyspnea.**—We have to designate that form of dyspnea as physiological which results when the respiratory center is supplied with blood which contains less than the normal quantity of O or an increased amount of CO_2. In the clinic it is difficult to give an absolute definition, because the perceptions of objective and subjective dyspnea (that is, which are only present in the sensations of the patient), as well as the dyspnea with and without deficiency of oxygen or of blood overladen with carbonic acid, are much mixed. Generally the clinician speaks of objective dyspnea in the following cases: if the respiration is labored, whether the number of respirations be normal, or prolonged, or more frequent. Finally, in all cases of

increased respiration, if rapid and labored breathing are combined, dyspnea is caused by all those conditions that interfere in any way with the exchange of gases in the lungs.[1] But there is another condition which manifests itself by an increased formation and giving off of CO_2; that condition is fever.

Labored respiration with normal or diminished frequency takes place in stenosis of the upper air-passage—that is, of the larynx and trachea. Intratracheal tumors, foreign bodies, inflammations (especially croup), cicatricial strictures (generally syphilitic), granulations, also compression from without, and lastly paralyses of certain laryngeal muscles[2] which produce narrowing of the air-passage. The slow and labored respiration in these cases seems a perfectly intelligible means of satisfying the requirements for oxygen, notwithstanding the fact that it is more difficult for air to enter.

Strictly speaking, this form of dyspnea often occurs in diseases of the brain.[3] At the acme of respiration in Cheyne-Stokes breathing we must speak, too, of there being dyspnea.

Increased Frequency of Respiration Occurs—

(*a*) In *fever.* Here it is often simply increased frequency, the breaths being deeper, but sometimes also we notice that they become somewhat labored (without its being a question of complication of the thoracic organs). The amount of quickening of the respiration varies very much according to the nature of the disease and with the individual. Nervous persons often breathe remarkably rapidly in fever; with children respirations as high as sixty or more to the minute have often been observed. Nevertheless, in fever every case of marked increase in frequency of breathing must lead to an especially careful examination of the thoracic organs. The cause of fever-dyspnea is, moreover, not alone the increased formation of CO_2, but is also the result of the irritation of the respiratory center by the warmer blood, as has been proved upon animals by an artificial increase of the temperature of the body. Finally, as a third cause of fever a direct effect of toxins upon the respiratory center is not excluded.

Fever-dyspnea may be increased by association with that caused by diseases of the respiratory apparatus.

(*b*) In *all conditions that are connected with pain in breathing.* Here belong all diseases of the pleura or of the lungs in connection with the pleura (especially croupous pneumonia), inflammatory affections of the diaphragm (trichinosis), of the peritoneum (especially the diaphragmatic peritoneum), fracture of ribs, and severe rheumatism of the muscles of the thorax.

Rightly to explain this form of dyspnea is often of the greatest therapeutic value; it may sometimes (not always) be relieved by a narcotic.

(*c*) In *diseases of the bronchial tubes* which narrow or close the tubes by the secretion or exudation. Here belong all forms of bronchitis and also bronchial asthma. In the latter disease there is much less swelling and exudation than from bronchial spasm of neurotic origin, which chiefly causes the dyspnea. No doubt spasm of the

[1] See under Cyanosis.

[2] See under Inspiratory Dyspnea.

[3] Also see page 81.

diaphragm is associated with this sometimes, which causes a prolonged inspiratory expansion of the lungs, and of course this increases the dyspnea.

Where there are bronchial asthma and croupous bronchitis in addition to laryngeal croup, there is generally very severe dyspnea with quicker and very forced respiration. Simple catarrh of the bronchial tubes generally leads to quickening of the respiration without the breaths being deeper; for a complete closure of the bronchial tubes cuts off a large section of lung, and so breathing is entirely lost in this section, as in capillary bronchitis, especially in children. The consideration of this condition properly belongs to the next section, in that it results in the lung-tissue itself becoming diseased.

(*d*) In all conditions in which the breathing surface of the lungs is diminished or the volumetric variation of the lungs, which is necessary for respiration, is disturbed. These are—

All *diseases of the lungs:* the different forms of pneumonia, edema of the lungs, infarction, tuberculosis, emphysema (this not only on account of the diminished breathing surface, but also the loss of elasticity, and hence diminished contraction of the lungs during expiration); the different forms of *pleurisy with exudation, pneumothorax; tumors in the chest-cavity* which diminish its capacity; *abdominal affections* which push up the diaphragm;[1] marked *kyphoscoliosis*, with the resulting deformity of the chest and consequent unfavorable condition for breathing; *paralysis* of the muscles of respiration; and also *tonic and clonic spasm* of the muscles of the chest, as in tetanus and epilepsy, which may occasion the most severe dyspnea.

As is evident, these diseases differ widely from one another. Those that diminish the chest-cavity, if they are inconsiderable, sometimes merely restrict the inspiratory expansion of the chest, and so affect the lungs; but if they are marked, then they directly compress the lungs and hence diminish their breathing-surface.

It has been already stated that in a number of these conditions the need of oxygen may be met by a substitution of diaphragmatic breathing in place of the diminished costal breathing, and *vice versâ*. It is, of course, very calamitous when there is a combination of several causes of dyspnea, as, for example, when a subject of kyphoscoliosis has an abdominal affection which presses up the diaphragm or has inflammation of the lungs.

Accommodation, adaptation, plays an important part in many chronic diseases which occasion dyspnea. This becomes most strikingly evident if we compare the terrible dyspnea of beginning pneumothorax with the relatively comfortable condition of patients who have continually at their disposal for breathing only one lung or even only a part of a lung. In many of these cases it is easy to understand this accommodation—chronic cases, especially phthisical patients, who here come prominently into view, are generally anemic, and therefore require, at least when quiet, only a very small interchange of gases in the lungs; but, nevertheless, every effort at muscular exertion immediately causes dyspnea. On the other hand, "lung-dyspnea" is generally considerably increased by the fever which accompanies an acute

[1] See above.

disease. Likewise, there are cases where we cannot dispense with the idea, which formerly was not clear, of an "accommodation."

Dyspnea further occurs—

(*c*) In diseases of the heart which cause stasis of blood in the lung-circulation. These are insufficiency or stenosis of the left auriculo-ventricular opening; also heart-failure, which may occur in all diseases of the heart.

Here the dyspnea is partly explained by the defective aëration of the blood in consequence of the slower circulation in the pulmonary capillaries. But that is not the chief factor, particularly in compensated mitral defects. The question here is regarding another element, which seems to have been made clear by von Basch. In consequence of the overfilling of the pulmonary vessels with blood the lungs are enlarged in volume, and they also contain more air, but at the expense of their elasticity. They become rigid—that is, they are capable of only slight alterations in their volume, similar to emphysematous lungs, but from an entirely different reason. This swelling and rigidity of the lungs, according to von Basch, gives the first satisfactory explanation of the peculiar dyspnea of compensated mitral defects.

Increased and forced respiration. Forced respiration may at any time be associated with rapid breathing by increase of dyspnea. The only exceptions to this are those cases that arise from pain and paralysis, both from reasons that are easily intelligible.

Mechanism of forced respiration. This is, in the most characteristic way, different from normal breathing—namely, that while the muscles of ordinary inspiration and the mechanical conditions of expiration no longer suffice, inspiration and expiration are assisted by the action of the auxiliary muscles of respiration.

The auxiliary muscles of *inspiration* are—the scaleni muscles in the male (in the female they act even in quiet breathing) as elevators of the first two ribs; the sterno-mastoidei draw up the sternum when the head is fixed; the pectoralis major and minor, the levatores costarum, the serratus post. super., all of which act as elevators of the ribs, the first named when the upper arms are fixed. In more severe dyspnea the trapezius, the levator scapulæ, the rhomboideus are brought into action to elevate the scapula; in severest dyspnea the extensors of the neck assist also, and then we notice the extension of the alæ nasi;[1] when the mouth is open the soft palate is seen to be drawn up during inspiration; and, finally, even those muscles that dilate the mouth and depress the larynx may be brought into action.

The muscles have very varying degrees of importance, the greatest being the work of lifting up the ribs, the sternum, and the shoulders. The expansion of the alæ nasi as a symptom is not unimportant, but really does not at all assist in breathing.

In *expiration* the following muscles act in assisting respiration: Of first importance are the broad muscles of the abdomen, especially the transversalis, which compress the abdominal contents, thus pressing up the diaphragm; further, the quadratus lumborum and serratus post. infer., which draw down the lower ribs.

It is easy to distinguish at a glance the moderate drawing-in of the

[1] See under Nose.

thorax and epigastrium which occurs in normal passive expiration from the active expiration of dyspnea, by the energy of the act in consequence of muscular contraction. Moreover, the contraction of the broad muscles of the abdomen is plainly to be seen.

Patients with *forced respiration* exhibit still other appearances which partly stand in direct relation to the increased energy of the breathing.

That the thorax may be entirely easy and that the auxiliary muscles may be able to act better, patients prefer the upright posture to lying down.[1] Indeed, in very severe dyspnea they may not be able to lie down at all; the arms are steadied, in order that the upper arms and shoulders may furnish a fixed point for the auxiliary muscles; and in order that the sterno-cleido-mastoidei may act most efficiently in assisting respiration the neck is stretched and the face somewhat elevated.

Not infrequently the breathing is audible; in forced respiration it is panting, groaning. In stenosis of the larynx or trachea we hear the before-mentioned hissing—*stridor laryngeus vel trachealis.* The voice is weak, often suppressed; the patient speaks with short, unnatural pauses—interrupted or broken speech.

Here belongs the so-called inspiratory "drawing-in." Even in healthy people we sometimes notice with forced respiration that the lower intercostal spaces in the beginning of inspiration sink in somewhat, instead of, as in healthy persons, a simple flattening-out from the contraction of the intercostal muscles. Drawing-in that is more marked and is prolonged during the whole of inspiration under all circumstances is pathological; with the very yielding thorax of children even the ribs and the lower part of the sternum may share in the condition. It shows that the lungs do not follow the motion of the thorax—that, therefore, the air is prevented from entering the alveoli.

Hence, all forms of *stenosis of the larynx*, of the trachea, and likewise the rare stenoses of the two primary bronchi, cause inspiratory drawing-in on both sides, most markedly of the lower part of the sternum, the lower ribs, and intercostal spaces; if the stenosis is very marked, the condition is extended to the upper ribs and intercostal spaces as far as the jugular and supraclavicular spaces. But *stenosis of only one bronchus* causes inspiratory drawing-in of one side when the breathing has a certain degree of force, besides "lagging" of the affected side. *Bronchitis of the smaller tubes*, especially in children, may occasion inspiratory drawing-in in a more circumscribed way, as only the lower part upon one side. But we may also sometimes see an extended, very marked drawing-in with extensive capillary bronchitis (with atelectasis, broncho-pneumonia) in children.

As regards frequency, laryngeal croup and capillary bronchitis in children take first place among the causes of inspiratory drawing-in.

There are two reasons why stenosis of the upper air-passage causes the drawing-in to be greatest at the lower part of the chest, and which may also affect the ribs of this part: first, the air entering the lungs reaches the lowest part, as being the farthest removed, last; secondly, if the thorax is yielding, it is drawn in by the contraction of the diaphragm, for if the diaphragm cannot descend when it contracts, since

[1] See Orthopnea, p. 29.

the lung does not follow it, then the dome of the diaphragm becomes a fixed point, and the thorax in the neighborhood of the insertion of the diaphragm is drawn inward and upward. Moreover, the lateral region of the thorax above the insertion of the diaphragm sinks in so much because the thorax is softest, and also because there is frequently here the greatest difference between the external and internal pressure.

Also, expiratory bulging sometimes takes place in the supraclavicular depression,[1] especially in marked emphysema of the upper part of the lung, as, for example, after whooping-cough; or in the upper intercostal spaces when large cavities are adherent to the chest-wall, as in pulmonary phthisis. With this condition there is a strongly-marked pressure in the thorax; hence it is observed only in very forced expiration, and especially in strained coughing.

Very frequently we find in cases of lung-cavities with expiratory bulging—especially frequent in the second intercostal space—the affected intercostal muscles very much shrunken, sometimes fatty degeneration of them.

Finally, the picture of such an unfortunate will be completed by the expression of subjective anxiety, sometimes of the most fearful agony; by the peculiar expression of the eyes, which is caused by the dilatation of the pupils which usually accompanies dyspnea, with occasional protrusion of the eyeballs;[2] by the cyanosis and frequent cold sweat[3]

According as inspiration or expiration, or both, are difficult, or the auxiliary muscles of respiration are brought into action, we distinguish an *inspiratory* (pure or preponderating), an *expiratory* (pure or preponderating), a *mixed dyspnea.*

Purely inspiratory dyspnea exists with paralysis of the posterior crico-arytenoid muscles (dilators of the glottis): here expiration is free, since the escaping current of air presses the vocal bands apart; on the other hand, the in-rushing air brings them in contact like valves, and hence inspiration may be hindered even to threatened suffocation. *Tumors and foreign bodies* may, moreover, be sometimes so located as, by valve-like closure, almost completely to preclude inspiration. Further, inspiratory dyspnea occurs with increased activity of other muscles when certain respiratory muscles are paralyzed[4] (as, for example, in paralysis of the diaphragm there is increased thoracic breathing, with co-operation of the auxiliary muscles).

Purely expiratory dyspnea is observed with movable tumors situated below the glottis: the outgoing air pushes them against the rima glottidis, but in expiration they are drawn to one side.

Moreover, a *preponderating expiratory dyspnea* is peculiar to *bronchial asthma* (in addition to the always present inspiratory). Probably we correctly assume that the smallest tubes, spasmodically narrowed, are still more compressed by the pressure in the thorax during expiration.

The disease that most frequently causes expiratory dyspnea is emphysema of the *substance of the lungs;* the diminished power of expiration is chiefly from the diminished elasticity of the lung-tissue, the contracting force of the lungs; generally there is, besides, dimin-

[1] See p. 68. [2] See Nervous System. [3] *q. v.* [4] See p. 85.

ished thoracic breathing, since, if the thorax be too rigid to expand during inspiration, then it is also not contracted either by virtue of its own elasticity or the traction of the lungs.

Bronchial asthma of long duration always causes emphysema of the lungs; then, of course, there is a twofold cause of expiratory dyspnea.

In genuine emphysema of the lungs there is always also well-marked inspiratory dyspnea on account of the atrophy of lung-tissue and capillaries of the lung, and hence diminished breathing-surface. Moreover, it will be understood that whenever there is expiratory dyspnea, if the difficulty of expiration is not equalized by forced or prolonged expiration, there must result a simultaneous inspiratory dyspnea; there is a diminished interchange of gases in the lungs resulting from the incompleteness of the act of expiration; there is a demand for oxygen, and hence forced inspiration. There is no expiratory dyspnea with vicarious emphysema of the lungs.

Mixed dyspnea—that is where it is manifest in equal degree in inspiration and expiration—is by far the most frequent. It accompanies all the diseases of the respiratory organs not mentioned here; also diseases of the heart, and fever.

Palpation of the Thorax.

This method of examination has, on the one hand, an independent value, and on the other it confirms, and with sufficient practice even adds to, the results of inspection. It is, therefore, very wrong to omit it. It is indispensable on account of its simplicity, and because, like inspection, it quickly furnishes a result in a general way; moreover, its result is often decisive in differential diagnosis, in a certain direction, relative to vocal fremitus.

Palpation of the thorax with reference to the respiratory organs is made for the purpose of ascertaining—

1. Possible pain upon pressure.
2. The respiratory movements of the thorax, especially as to symmetry.
3. Any friction-sounds or râles that may be felt.
4. Vocal fremitus.

In addition, there are some rare conditions that are not unimportant in differential diagnosis.

The examination with reference to the first and second points may be combined with inspection; the trial of the third point may suitably be settled during auscultation, either before or after. Ordinarily, we test the *vocal fremitus* after the completion of percussion and auscultation; hence we conclude the physical examination of the thoracic organs by noticing the vocal fremitus.

We pause here in the course of the examination, and only speak of the first and second points; the two others will be introduced under the heads of Percussion and Auscultation.

1. Pain caused by Pressure upon the Thorax.—In diseases of the chest pain is common, accompanying the diseases or elicited by pressure. In case it really refers to an internal organ, and not to the

chest-wall, it indicates disease of the pleura or complication with the pleura. By carefully feeling the intercostal spaces with the tips of the fingers the region that is tender on pressure may be very exactly defined; it is generally less extensive than the territory of spontaneous pain, since the latter ordinarily "radiates."

This tenderness sometimes exists with exudative pleuritis, but in this disease it is often wanting; more frequently it is seen in croupous pneumonia which also involves the pleura, and also in phthisis. In the latter disease it generally depends upon callous thickening of the pleura.

It is very important, but also frequently difficult, to distinguish between pleuritic pains produced by pressure from those arising in the *soft parts of the chest-wall* or the *ribs*. Phlegmonous inflammations and abscesses of the chest are, of course, easily recognized. Pain proceeding from a rib is generally characteristic: quite circumscribed, it occurs only when pressure is made upon the affected rib (caries, periostitis, over fractured ribs, slight pressure); also, rheumatism of the chest-muscles occasions no great difficulty, at least when it is in the superficial muscles: the muscle is ordinarily sensitive if pressed between two fingers. On the other hand, it is often not easy to distinguish between pleuritic pain and *intercostal neuralgia;* the latter can sometimes be distinguished by Valleix's points of tenderness, which stand wholly out of relation to deep breathing or cough.[1] It is important to remember that neuralgic intercostal pain may be present in affections of the pleura, as in tubercular thickening of the pleura in the lower part of the thorax.

In short, we ought, in the absence of other indications which point to a disease of the internal thoracic organs, to refer a pain produced by pressure upon the thorax rather to something else than to the pleura; only continuous pain, always at the same places, over the upper sections of the lungs, arising either spontaneously or from pressure, is suspicious; this may indicate irritation of the pleura from *tuberculosis of the apices*.

Fractures of the ribs are recognized by crepitation and by dislocation of the fragments; also often by the fact that pressure at any part of the broken rib causes pain at the seat of fracture. Moreover, fracture of the rib may cause pleurisy. *Caries of the rib* may also excite pleurisy. Then in recognized pleurisy caries may be proved to be the cause by the circumscribed pain elicited by pressure upon the rib.

It must also be mentioned that if a purulent pleuritis breaks outward (*empyema necessitatis*) it causes peripleural inflammation, and with this there is pain upon the slightest pressure, besides swelling, redness, heat, edema of the skin, and, lastly, fluctuation.

To the above-mentioned conditions revealed by palpation of the thorax must be added *pulsations of the heart* felt through a portion of infiltrated lung lying over the heart, and also in the so-called *empyema pulsans* (empyema pulsatile). This occurs when there is an accumulation of pus lying over the heart, almost always upon the left side, to which the pulsation of the heart is communicated. In some cases it is

[1] See Nervous System.

very difficult to distinguish it from aneurysm of the aorta. It can only be done by taking a comprehensive view of the case. (We must be on our guard in puncturing or in making an exploratory puncture.) Sometimes pulsations are even found on the left lower posterior portion of the thorax. Usually several causes combine to produce the pulsation: paresis of the intercostal muscles, higher pressure of the exudate, direct contact with the heart, lastly, as indispensably necessary, powerful action of the heart.

2. Testing the Movement during Respiration.—With special reference to symmetry, with some practice, palpation is a most excellent method. It gives more exact results than inspection, and makes the further examination easier, in that it directs the attention immediately to the diseased side or the region of the thorax affected.

The respiration is examined by placing the two hands alike upon the two sides of the chest. In order to test the breathing of the upper divisions of the lungs, place the hands flat in front, gradually diverging below, so that the tips of the fingers reach to the lower border of the clavicle. For examining the lower parts spread out the hands with the thumbs extended, so that the thumbs rest upon the angle of the ribs, and the fingers toward the sides of the thorax. Behind, only the respiration of the lower portion of the chest will be tested by laying the flattened hands, with the thumbs extended, upon the surface in such a way that the points of the fingers reach about to the middle axillary lines.

For exact examination it is necessary, if possible, for the physician to be directly before or behind his patient; the latter position especially is often difficult when the patient sits in bed; it is best, then, to have the patient slide somewhat down toward the foot of the bed. It must also be remarked that when the patient is lying down there is not infrequently produced a one-sided after-drawing in front and above by an imperceptible inequality in the position of the patient. It is, however, usually best to look out for this symptom in front above when the patient is in an upright position.

When palpation is well performed, "lagging" over the apex in beginning phthisis or the "lagging" of the lower part of one side in pneumonia, pleurisy, infarction, etc. is recognized with great exactness. This is of great importance, because, as I have already said, "lagging" may be in many diseases for some time the only symptom.

We may also test the contraction of the diaphragm with reference to its symmetry by palpation. We place the hands so that the fingertips cover the epigastrium; in this way may be detected the lack of contraction upon one side (*pleuritis diaphragmatica*, local peritonitis, unilateral paralysis of the phrenic nerve). Failure to contract upon both sides is, of course, seen at once.[1]

Benczúr and Jonás[2] have lately tried to use certain differences in the temperature of different parts of the surface of the body for a systematic demarcation of organs which lie against the parietes of the body, especially of the lungs, from parts which do not contain air. By passing the volar or dorsal surface of the fingers over the thorax they have found that the region over the lungs was always warmer, and

[1] Compare p. 74.

[2] *Deutsch. Archiv f. klin. Medicin*, Bd. xlvi.

they assert that by means of thermopalpation it is possible to make out exactly the line of demarcation between the lungs, heart, liver, etc., also from pleuritic exudations, and even to make out their relative dulness. That there are differences of temperature they prove by means of delicate methods of measuring. However, after having made a few experiments ourselves, we are obliged to deny the clinical usefulness of thermopalpation, because the respective differences are too slight to be indubitably recognized by the finger.

PERCUSSION OF THE THORAX.

General and Preliminary Remarks regarding Percussion.[1]

In daily life we learn on every hand that bodies of different physical structure give forth different sounds when struck. We also sometimes strike an object in order to determine from the sound it gives forth what its physical condition is—that is, whether it is hollow or solid. This is the principle upon which percussion is practised on the human body: from the sound elicited by the blow we judge of the physical condition of the part which lies beneath the covering of the body within the sphere of our percussion-stroke.

Hence, percussion gives direct information regarding organs or parts of organs which lie approximately near to the surface of the body; in general, by this method we penetrate only to the depth of 5 or, at most, 7 cm.

1. History and Methods.—The honor of the discovery of percussion belongs to a physician of Vienna named Auenbrugger; the paper in which he made known his method appeared in 1761 under the title, *Inventum novum ex percussione thoracis humani ut signo abstrusos interni pectoris morbos detegendi.* For almost half a century Auenbrugger's discovery was, on the one hand, declared to be without importance, and, on the other, was ridiculed until the year 1808, when Corvisart, body-physician to Napoleon I., emphatically revived and largely improved it by a translation into French, with a commentary. Then the truth began really to prevail, especially by the influence of Piorry in France and Skoda in Vienna. The former was the founder of *topographical percussion.* During fifty years the method gradually became common professional property. Further, and up to the most recent time, it experienced improvement and explanation of every kind, especially by Wintrich, Traube, Biermer, Gerhardt, and Weil. For some time past, especially since the labors of Weil, it appears that a degree of certainty has been reached in regard to this proceeding.

In the course of the development of percussion several methods of striking the body have been discovered, most of which still have value to-day.

Auenbrugger struck directly upon the thorax with the tips of the fingers—*direct* or *immediate percussion.*

Piorry discovered *indirect* or *mediate percussion* in that he placed under the percussing finger a small plate of ivory, a *pleximeter.*

[1] In this chapter the author follows in many ways, but not entirely, the views and methods of presentation of Weil, whose well-known and excellent work has done much to establish this subject upon an accepted basis.

Wintrich introduced the percussion-hammer, which had already been sometimes used by Laënnec and Piorry, in place of striking with the fingers.

But finally, in more recent times, the method of indirect percussion, without instruments, has very widely prevailed. The index or middle finger of the left hand is used as the plexímeter, which is placed upon the spot to be percussed, and it is struck with the index or middle finger of the right hand (finger-percussion).

Of these methods, that of Auenbrugger, the direct, has been dropped as being less practical, while nowadays the three in use are all examples of the indirect method:

I. Finger-percussion.
II. Finger-plexímeter percussion.
III. Hammer-plexímeter percussion.

All three are practised and taught by good teachers of percussion; all three, in reality, yield equally exact results; the secret of their value lies in *their application.* The fact of the matter is that one who thoroughly understands finger-percussion can very quickly acquire a knowledge of the two other methods. Hence I am most heartily in accord with those who in their teachings and writings emphatically recommend their students at first to practise the finger method of percussion exclusively.

I think it superfluous for me here to go into particulars regarding the *technique*—these can only be made clear in the clinic—but I must remark that the greatest difficulty in finger-percussion is in holding the percussing finger crooked like a hammer, and at the same time having the wrist-joint move quite freely. Also, the numerous forms of percussion-hammers and plexímeters (the latter of glass, ivory, hard rubber, and wood in different forms) cannot be described here. It appears to me that the hammer with a wooden handle and a metal head, not too heavy, is rather to be recommended; likewise, a medium-sized oblong ivory plexímeter, about 2 cm. wide, and the so-called double plexímeter of Seitz. Even to those who practise finger-percussion this last is recommended for percussing the supraclavicular depressions.

There have lately been invented small thimble-like coverings for the percussing finger to be used as a substitute for the hammer. They seem to us to be worthy of notice, though we have not yet had much experience with them. But after various trials we do not think it is practical to provide the finger that is used as a plexímeter with a rubber ring or anything of that sort.

There is one point of great importance—that the individual should, as much as possible, be similar and uniform in his methods throughout: in percussing, if the finger method is used, he should always strike upon the index or always upon the middle finger of the left hand; the plexímeter, if that is used, should always be used in exactly the same way, etc. Nothing is worse than frequently to change methods or instruments, be the change ever so slight. But if physicians, as is true of many, are accustomed ordinarily to percuss without an instrument, but at certain parts of the thorax where it is difficult to use finger-percussion they regularly employ a plexímeter

or both pleximeter and hammer, there is no objection to this twofold method; only the examiner must be master of the two methods which he employs. It is well also always to repeat the same method upon the same parts of the body.

2. Qualities of Sounds.—By striking upon the body we cause a sound. This percussion-sound differs according to the condition of the part of the body which is shaken by our percussion-blow. From this fact there results directly two main points, which form the basis of the doctrine of percussion:

I. When we strike upon a solid portion of the body entirely free from air we elicit a toneless sound of the least possible intensity and duration; it is designated as "absolutely deadened" or as a "thigh sound," since it is like that caused by striking upon the thigh. [Deadness: I have frequently used this word and its derivations as giving a useful and accurate discrimination from the familiar English terms flatness, dulness. Deadness is more than dulness.—*Translator.*]

II. If organs containing air lie in the range of our percussion-blow, then these give forth a sound of a certain intensity, duration, and tone; this sound is designated as "clear."

The clear sound of organs containing air may have only a different degree of intensity or clearness. Its intensity depends upon—

1. The *length of the oscillation.* It is therefore stronger the stronger the blow, and, moreover, the nearer the organ containing the air is to the percussing finger—that is to say, the less the percussion-stroke is weakened by the tissue, as fat, muscles, bones [also clothing], intervening between it and the air-cavity.

2. By the volume of the parts of the air-containing tissue set in motion.

Hence, with equal strength of percussion we have in different parts of the body different *intensity* and different *clearness* of sound according to the greater or less amount of air which the tissues contain, or according to the nearness or distance of the air-cavity from the surface of the body—that is, from the percussing finger.

It is according to the change of these conditions in the human body that we obtain the different clear sounds: we may meet every grade from absolute deadness to a very clear—the peculiarly clear—sound. These intervening grades are designated as "relative dulness" (that is, in comparison with a real clear sound it is dull).

Absolutely dead or dull sounds differ according as they proceed from muscle, bone, etc. We cannot wholly ignore these differences as if not existing.

On the other hand, the clear sounds fall into the two following important divisions:

1. *Tympanitic sound* (the name is from tympanon; the kettle-drum or tymbal, not exactly, but very nearly, produces it). This approaches a musical note, so that we can exactly define its place on the musical scale, and it is actually shown formed from regular oscillations in the rotating reflected image of the sensitive gas-flame. It possesses also, according to the different conditions to be described later, sharply definable differences of pitch. A tympanitic sound, such as is frequently met with in the body, can easily be produced if one strikes

upon his own cheeks which have been inflated, but not too strongly stretched.

2. The clear sound called *non-tympanitic*, also more briefly "lung-sound"—a very practical designation. This has no sound definable by its pitch, but yet it may be known in general as "high" or "deep."

Hence, both the tympanitic and the non-tympanitic sounds have a certain *intensity* and *duration;* but while the latter may be only approximatively designated as high or deep, the pitch of the tone brings it toward the tympanitic. Both occur in a very high degree of clearness and in all degrees of relative dulness ("relative dulness" or "dull tympanitic sound"), even to an often unnoticeable transition to absolute dulness.

1. We give here and later on some schematic drawings which are designed to facilitate the understanding of the foregoing points. We can recommend the application of this manner of representation to teachers of percussion, as well as to the student for his private studies or for his notes of what he has seen and heard in the clinical courses.

Percussion of the lungs is represented in a rough schematic drawing in which a long arrow signifies strong percussion, a short one weak percussion. In the first and third drawings the parietes, for the sake of simplicity, are represented as being of uniform thickness. The hatched triangles in each figure represents the portion of the lung

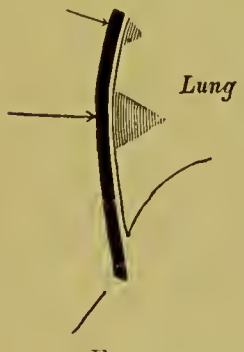


FIG. 25.

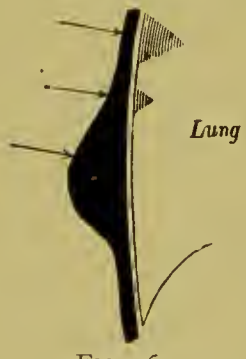


FIG. 26.

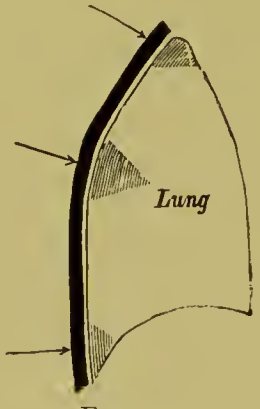


FIG. 27.

FIG. 25.—Schematic representation of the difference between strong and weak percussion, the conditions being otherwise the same. The length of the arrow indicates the strength of the percussion, the size of the hatched triangle, the extent of lung-tissue affected by the percussion-blow, and also the intensity of the sound.

FIG. 26.—Represents the different results, with equal percussion force, where the thickness of covering varies: clear sound; relative dulness; no sound—that is, absolute deadened sound.

FIG. 27.—Represents the influence of the volumes of resonant body upon percussion: over the apex and border, on account of the small volume of lung: with equal and moderately strong percussion and equal thickness of covering, the sound is less intense than over other parts of the lung.

which is set in vibration by the percussion-blow, and also the intensity of the sound. Figs. 25, 26, 27 show how the intensity of the sound is influenced

(*a*) by percussion with different degrees of force,
(*b*) by different thicknesses of the parietes, and
(*c*) by the depth or volume of air contained in the organ.

2. In the foregoing we give those designations which in late years we have without exception employed in our instruction on percussion. Regarding the large number of other terms for qualities of sound which the older teachers of percussion have introduced, but which, to the great advantage of clearness of mutual understanding, have more and more disappeared from the literature of the subject, we refer to the classical work by Weil on *Topographical Percussion.* We have in fact, as will be seen, followed the nomenclature proposed by Weil, with only one exception; the term *dull sound* is avoided, and in place of it we have employed the expression (which, it is true, is somewhat circumstantial) "absolutely deadened sound," or "thigh-sound." This was done because, over and over, we found that pupils were reminded of the "dull sound of the kettle-drum," "dull roaring," etc., and hence were confused—in short, because the expression does not grammatically designate what is intended in teaching percussion. "Absolute deadened sound" is an expression which has this advantage—that to the beginner it is a new association of words; it cannot, therefore, so easily occasion confusion. Moreover, the expression always summons one to a more exact testing as to whether, at the particular place, there is really absolute or only relative dulness, and also it seems to us preferable, for every teacher of percussion knows how much this is needed —that, for instance, in percussing the lower part of the right mammillary line the so-called relative liver-dulness is spoken of as absolute deadness.

3. For the sake of brevity and clearness we also have really not gone into the many ideas and the manner of explaining them presented by others on this subject, which was formerly quite confused, and is even yet difficult to master. But we cannot abstain from citing here, by reason of their historical interest, the three fundamental sentences from Skoda:

(*a*) All fleshy parts not containing air (except tense membranes and filaments), also fluid accumulations, give an entirely dead and empty, scarcely distinguishable percussion-sound, which can be demonstrated by striking upon the thigh.

(*b*) Only bones and cartilage when directly struck give a peculiar sound.

(*c*) Every sound which we elicit by percussing the thorax and abdomen, and which differs from the sound of the thigh or bone, comes from air or gas in the chest or abdominal cavity.

4. The acoustic character of the *clear* and that of the *relative* or *absolutely dull sound* is clearest expressed if we say: the dull sound is a very slight noise of short duration; the clear, non-tympanitic sound is a noise louder and of longer duration, with a trace of being a note; this latter, however, is so little apparent that it either cannot at all be recognized, or only in general, as to its being high or deep. In the tympanitic sound, with the discordant mingling of tones, there predominates a tone of such a character that it is plainly heard and its musical pitch distinguished.

3. The Conditions that Determine the Quality of the Sounds and their Production in the Body.—The Feeling of Resistance.—The tympanitic sound exists—

1. Over cavities that contain air or gas if they are surrounded by walls moderately smooth and capable of reflexion, and if they communicate with the external air through an opening; the walls may be stiff or yielding. The intensity of the tympanitic sound thus produced depends upon the conditions (mentioned on page 94) influencing the intensity of clear sounds in general. The musical pitch of the sound is determined by—

(*a*) The *size of the communicating opening:* the larger it is, the higher the tone;

(*b*) The *volume of the cavity* containing the air: the larger the cavity, the deeper the tone;

(*c*) If the walls are yielding, membranous, by their *tension:* lax membranous walls make the tone deeper.

2. Over *air-containing cavities* with yielding, membranous walls if the cavities are closed—that is, do not communicate with the external air; only the walls, and with them the enclosed air, must not be too tense. Here the *pitch* is determined only—

(*a*) By the *volume* of the air-cavity: see above under 1. (*b*).

(*b*) By the degree of *tension of the wall:* see above under 1. (*c*).

But if the tension of the wall (and with it the enclosed air) of a closed cavity reaches a certain degree, then the percussion-tone becomes clear and non-tympanitic. Likewise, cavities that are closed on all sides by stiff walls give a non-tympanitic sound.

The tympanitic sound mentioned under 1 is called "open," that under 2 "closed." The former has a much more pronounced tympanitic character—that is, the pitch of the tone appears more distinctly—than the latter.

When the cavities are cylindrical, communicating outward by an opening, the pitch of the tone is determined by the length of the cylinder: the longer it is, the higher the tone. Some experiments, illustrating what has been said, are easily performed and are strongly recommended to beginners: Take an empty Florence flask and percuss upon its mouth, either directly or hold the pleximeter lightly over its mouth; then diminish the quantity of air by partly filling the bottle with water; if possible, also compare the differences of pitch which are produced by different lengths of the neck of the bottle, other conditions remaining the same. Percuss a rubber gas-bag which is at first only moderately inflated, then more tensely, with air. In this way one can very easily illustrate the most important of the laws that have been mentioned.

3. Finally, *tympanitic sound* occurs under quite other conditions—namely, in certain conditions of the lungs which have this in common, that they probably accompany a want of tension of the lung-tissue.

Referring to what was said above under 1, we add that the *open tympanitic sound* occurs in the human body, under normal relations, when the *mouth, larynx, and trachea* are percussed; pathologically, when percussing *lung-cavities* which are in open communication with the air-passages; further, if, in consequence of shrinking of the apices of the lungs, the trachea, or in consequence of shrinking or thickening of the lung where it covers a fissure, a primary bronchus would be reached by the percussion-stroke, and would, therefore, be itself per-

cussed; and, finally, the open tympanitic sound sometimes occurs with *open pneumothorax.*

Herewith we notice a peculiarity of this sound which truly stands in a certain (although still not altogether clear) relation to the laws above enunciated regarding the pitch of the open tympanitic sound: the sound is higher with the mouth open, deeper with the mouth closed. If this occurs when percussing a lung-cavity (or also in open pneumothorax), it is called *Wintrich's change of sound;* if on percussion of the trachea or a primary bronchus, then we speak of *Williams's tracheal tone.*[1]

In addition to what was said above under 2, we remark that in the human body the *closed tympanitic sound* is heard over the stomach and bowels; in rare cases over closed pneumothorax; and, finally, in pneumopericardium.

Now, while it is difficult to apply the rules regarding the change of pitch to the open tympanitic sound, since the cavities of which we are speaking are of extremely complicated form and have very different walls, the influence on the one side of the volume of the cavity, and on the other of the tension of a membranous wall, is shown over the stomach and intestines. A greater volume, as in the stomach and colon in comparison with that of the small intestine, deepens the sound, while increased tension heightens it, and even renders it non-tympanitic.

We add to what was said above under 3 that the normally clear, non-tympanitic sound over the lung *becomes tympanitic* if the tension of the lung-tissue diminishes—*i. e.* if the lung, following the pull of its elasticity, is able to retract. This is true in all cases where the pleural cavity is diminished, hence especially in *exudative pleuritis.* The tympanitic sound is found where the retracted lung lies against the thorax. All the other changes of the thoracic and abdominal cavities which have been mentioned before[2] as working in the same way, occasion these phenomena.

Probably, for the same reason—*i. e.* in consequence of the relaxation of the lung-tissue—a tympanitic sound is heard in *croupous pneumonia* during the stages of engorgement and of resolution; in *œdema of the lungs;* and finally in the neighborhood of thickened parts of the lungs. In the latter relation the tympanitic sound over the apices of the lungs in the beginning of tuberculosis, where lung-tissue containing air is situated between groups of small tubercular masses, is of some diagnostic importance.

In these cases we must assume that the lung-tissue has become loose and ductile, and has therefore lost its power of stretching. It has not yet been established that this explanation is correct.

Metallic Sound.—We thus designate such a variety of tympanitic sound by which a *metallic character*, produced by a very high overtone, either occurring with the sound itself, a peculiar *metallic tone*, or it is produced afterward, *metallic after-sounds.* The metallic sound exists over not too small, very smooth-walled, regular cavities, both open and closed. Hence, we find it sometimes over the *normal stomach, intestines*, and sometimes over *lung-cavities*, in *pneumothorax, pneu-*

[1] See, regarding this, pp. 106, 113.

[2] See p. 85.

mopericardium. It is best brought out in percussion with the so-called rod plexímeter or in percussion-auscultation (Heubner).[1]

The *clear non-tympanitic sound* occurs where, "within the sphere of action of acoustics, there is found tissue containing air, but whose capacity for vibration is more diminished than in those cases in which the tympanitic sound occurs."[2] It is heard over the normal lungs—a remarkable fact, since a lung that has been removed from the body, even if it is inflated to a volume corresponding with the condition during life, gives a sound that more nearly approaches the tympanitic than the non-tympanitic. Why a lung in the thorax loses wholly the tympanitic character of its sound is not entirely clear, but we cannot help thinking that, in some way or other, the chest-wall is the cause.

The *intensity of this lung-sound* is sufficiently explained by the rules given above; its *pitch*, only approximately recognizable, is chiefly influenced by the tension of the lung-tissue. We have mentioned above that retracted and relaxed lung-tissue gives a tympanitic sound; if the tension is only slightly diminished, then there is only a *very deep (and abnormally clear) non-tympanitic sound.* This occurs, also, in emphysema of the lungs, but sometimes in exudative pleurisy, and also in pneumonia in the air-containing, compressed, infiltrated adjacent sections of the lungs. The transition from the non-tympanitic to the tympanitic sound over the lungs may be thus summarized: According to the diminution of the normal tension of the healthy lungs, there takes place in the thorax a change of the clear non-tympanitic sound to an abnormally clear and deep, and in very marked relaxation to a tympanitic, sound.

To the above corresponds the fact that in very deep respiration, at the height of inspiration, at many points of the thorax, the respiratory sound is distinctly higher, while in deep expiration it is deeper ("change of respiratory sound"—Friedreich).

Moreover, we hear the lung-sound over the *stomach* and *bowels* if they are very much inflated with gas, where gas, as well as wall, is under marked tension; finally, when the walls of the cavities of the body are made tense by the *entrance of air* into them. This especially happens in most cases of *pneumothorax* (except that open pneumothorax frequently gives a tympanitic sound).[3]

The deadened sound. Absolutely deadened or thigh-sound is met with "if only structures that are free from air lie within the sphere where the percussion-stroke acts acoustically" (Weil). Since this, at best—*i. e.* with the strongest percussion—reaches only to the depth of 6 to 7 cm., and not so much as this in a lateral direction, therefore in case of only strong percussion absolutely deadened sound, after all, would be found where we percussed over *airless structure* of sufficient size if an organ containing air were not directly in contact with it. If we percuss still less strongly, we should, as a matter of course, the sooner receive an absolutely deadened sound.

In the human body we have next to consider the internal organs not containing air, called "parietal" if they lie in contact with the wall of the body; and also the *coverings* (subcutaneous fat, muscles, bones)

[1] See later.

[2] Weil: *Handbook of Topographical Percussion*, 2d ed., p. 35.

[3] See above.

if they are of sufficient magnitude. Thus, frequently, in the region where the heart is parietal, and, further, where the liver also is, even with strong percussion there is absolutely deadened sound. Not infrequently, however, especially over the heart, absolute deadening does not exist, since the structures containing air lying under or near by may be reached chiefly through transmission by the chest-wall,

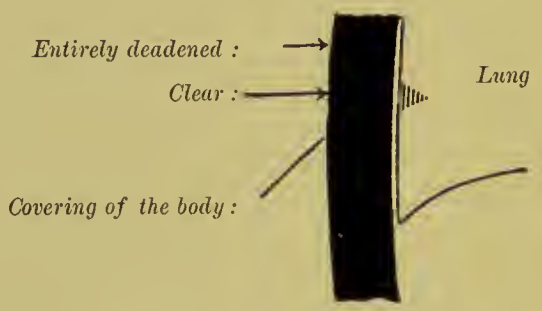


FIG. 28.—Diagrammatic representation of percussion over a thick covering of the body. The short arrow indicates weak, the long one strong, percussion. With weak percussion we have absolutely deadened resonance; with strong percussion a clear, although less intense, sound (indicated by the hatched triangle).

though it may be only by its vibration, and may give the clear sound belonging to the air-containing structures.

As regards the *skeletal coverings*, in abnormally fat persons and in edematous diseases, these sometimes attain such proportions that even strong percussion yields an absolutely deadened sound; in normal, moderately fat persons it is only the fossa infraspinata that very frequently gives an absolutely dull sound.

But, further, *parietal tumors*, and especially *fluid accumulations* in the pleura and peritoneum (more rarely *thickening of the lungs*), occasion absolutely deadened sound in case they, together with the skeletal covering, possess sufficient depth and breadth.

Moreover, over ribs markedly bowed, as over the point of sharpest bending-out of the thorax in kyphoscoliosis, absolutely deadened sound may take the place of the lung-sound; also here a peculiar change of the lung (aplasia) often plays some part. But under the circumstances mentioned above there may be relative or even absolute dulness of sound over perfectly normal lung-tissue.

Furthermore, it is to be remembered that when the body lies on pillows, etc., these tend to diminish sound in parts immediately in contact with them, because the integument and subjacent tissues, particularly the ribs, do not vibrate so readily when close against anything, and for the same reason they cannot transmit vibrations. Thus there is dulness in the sloping lateral parts of the thorax if the patient is lying upon his back in bed. This dulness, though insignificant, is yet pronounced enough to obliterate fine differences or to lead to error where a nicer distinction is required.

Relatively dull sound occurs where air-containing structures of only small size are percussed, or where structures containing air are made to vibrate only slightly by percussion, or where these two conditions are met with together. Thus, a relatively dull sound is obtained with feeble percussion of air-containing structures, while strong percussion

of the same yields a clear sound: the blow reaches only a small volume of the air-containing organ, and it moreover causes in it oscillations of only moderate amplitude. Likewise, where the volume of lung-tissue is small, as over the apices and just over the lower border of the lungs, the sound is relatively dull, and this is true even with strongest percussion, since there is here only a small portion of air-containing material to be acted upon. Finally, every layer of airless tissue which lies over an air-containing tissue or space causes a deadening of the percussion-sound of the latter—*i. e.* a relatively deadened sound—if the overlying layer is not so thick as to cause an absolutely deadened sound.[1] Subcutaneous fat, muscles, bones, parietal tumors, thickening of lungs, layers of fluid, callosities,—all these, as overlying airless masses, deaden the sound in proportion to their size.

A special description is required both of parietal and of *deeply-*



FIG. 29.—Diagrammatic representation of the value of gentle percussion in determining parietal condensation in the lungs.

The length of the arrow indicates the strength of the percussion, the size of the hatched triangle the extent of the vibrations in breadth and depth. We notice that weak percussion is better, because it gives a deadened sound over the thickening, while over the lung it gives a clear sound.

seated airless parts which normally contain air, such as occur especially in the lungs as *acute* and *chronic pneumonic thickenings*, *infarc-*

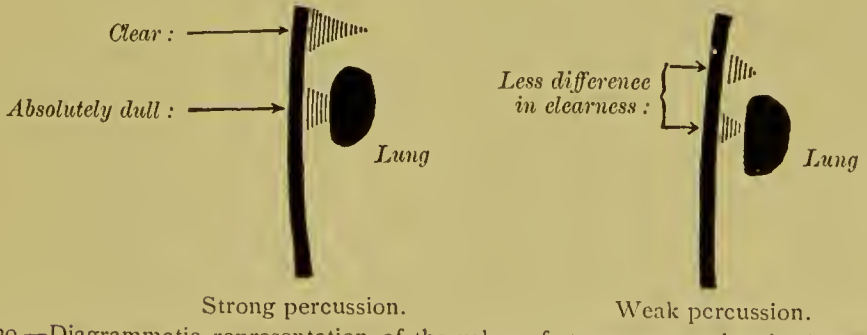


FIG. 30.—Diagrammatic representation of the value of strong percussion in determining condensation in the lungs lying at some distance from the surface.

The strength of the percussion-stroke is indicated by the length of the arrows. The hatched triangle shows the extent of the oscillations in breadth and depth.

tion, and *tumors*. For ascertaining such solidifications, if they are parietal, it is necessary not to percuss too strongly; then we shall

[1] See above.

plainly make out the place where there is air by the difference in sound if the given patch of thickening measures as much as about 5 cm. in breadth and 2 cm. in depth (see Fig. 29). Deposits which, on the other hand, are located at about 3 to 4 cm. in depth, if they are correspondingly large, may be detected, but only by very strong percussion; then we elicit a relatively deadened sound in the midst of what is quite normal, as is shown by Fig. 30.

Sensation of Resistance.—We introduce here the description of this symptom, although it really belongs under Palpation, but in truth it is most intimately connected with Percussion.

With the percussing finger (less distinctly with the hammer) the examiner forms an opinion of the *degree of resistance*, or, to express it better, concerning the *degree of capacity of the parts lying beneath it to vibrate*. This feeling of resistance is strongest, the power to vibrate least conceivable, where it is absolutely deadened, the sound identical with the "thigh-sound;" hence, normally, where we strike upon thick muscle, also bones and muscles; pathologically, it is especially distinct over *large pleuritic exudations*, *very thick pleura*, *solid parietal tumors of the chest;* over *large solid abdominal swellings;* and in extremely rare cases in *extensive thickening of lungs*, where the bronchi are completely stopped (as in the so-called "*massive pneumonia*" of the French).

When the *percussing hammer* is used to ascertain the feeling of resistance the index finger is placed upon the head of the hammer. This has always seemed to me a very poor substitute for finger-percussion.

Other authors, as Weil, find a marked feeling of resistance only over massive layers of fluid. I have often convinced myself of the presence of marked resistance in the cases above mentioned.

4. Topographical Percussion: Determining the Parietal Boundaries of Organs.—Only of a part of the internal organs can we determine the boundaries by percussion on the surface of the body. The conditions of such determinations are these:

(*a*) That the given organ be parietal.

(*b*) That it yield a sound differing from its surrounding tissues.

Hence, we can mark off the boundaries of a parietal organ that gives an absolutely deadened sound from one that gives a clear (tympanitic or non-tympanitic) sound, as the liver from the lung or stomach, the heart from the lung; of a parietal organ that gives a tympanitic sound from one that yields a non-tympanitic sound, as the lung from the stomach or the intestine; of parietal organs with tympanitic sounds of different pitch, as the stomach from the intestines; and also, though very seldom, two organs of non-tympanitic sound in case they are of very different pitch, as pneumothorax from lung lying against the opposite side.

But we can never recognize the boundaries between two organs giving deadened sound (heart and liver), nor between the heart and fluid effusion in the pleura.[1]

Method of Determining the Boundary.—Generally we percuss from an organ that yields a clear sound toward that which gives a deadened

[1] See below.

sound, and upon the line which stands perpendicular to the expected boundary-line. Hence the pleximeter or the pleximeter finger is placed parallel to where the expected boundary-line lies. We proceed by long stages upon this perpendicular (striking it at intervals of about 3 cm.), until the sound has so distinctly changed that we are convinced that we are over another organ. Then we define the boundaries by placing the pleximeter at shorter and shorter intervals until we have defined the boundaries as sharply as possible. This is traced by means of a blue pencil. After the boundaries have been determined at various points and they have been thus marked, then the points are united in a line, which is the boundary-line of the particular organ.

The rule most important to observe is to percuss very lightly along the border of the organ we are trying to locate. It is easy to see the reason for this: 1. *By strong percussion*, as of the liver close to the lower border of the lungs, we should at the same time disturb the adjacent lung, and so would elicit a noticeable clear sound, and we should then easily think that we were still over the lung. In the same way, in determining the lower border of the liver, by strong percussion we disturb the intestine which here lies under the thin portion of the liver, and so get a tympanitic tone. 2. The ear perceives the very slight differences of sound which exist upon the border-line (we remember the lower border of the lung, how the clear sound yielded by it must have slight intensity) better if the sound is itself slight.

For those who are trained the simplest method may be recommended, that on approaching the boundary between the two organs one should successively percuss the more lightly.

The dermatograph of Johann Faber for marking the boundaries on the body can be very strongly recommended.

In important cases it is advisable to mark upon an outline drawing of the body what has been found by percussion. One can use with great advantage rubber stamps for these outlines. They enable the physician to quickly enter his findings at the appropriate place in each individual history.

After this indispensable explanation of the general rules for percussion and their practical value, we again take up in succession the methods of examination of the respiratory organs, beginning with the percussion of the thorax.

Percussion of the Thorax, especially of the Lungs.

1. Methods.—It is best first to percuss patients who are out of bed in the standing posture, and later, if necessary for the front of the chest, lying down. Upon bedridden patients the examination of the chest is conducted with the patient in the dorsal position; for percussing the back we have the patient sit up. We must then take care that the patient sits in a symmetrical position, but with the least possible tension of muscles; the head is held exactly straight, and especially in percussing the supraclavicular depressions it must not be turned; in the dorsal position the arms lie quietly by the side of the thorax. Both in sitting and standing the patient bows the back a little, inclines the head slightly forward, allows the shoulders to hang, and folds the

forearms across the chest. Every contracting muscle increases the thickness of the covering by its swelling and increases the impression of dulness; hence, contraction of the muscles of the thorax must as much as possible be prevented.

In finger-percussion of the front of the chest with the patient in the dorsal position we approach the bed if possible so as to stand on the left side of the patient. From the other side it is not possible to place the finger of the left hand, used as a pleximeter, symmetrically[1] upon the two sides in both supraclavicular spaces.

We proceed in such a way as to compare at every situation the percussion-note of points that are symmetrically located. We must take particular care to strike exactly upon symmetrical points, otherwise the "comparative percussion" has no value. Moreover, since we wish to make an exact comparison throughout, we take care also not only to percuss at symmetrical points, but to percuss with equal strength and somewhat moderately.

We first percuss the supraclavicular depressions—first on the right, then on the left, whereby, in cases where it is of special importance, we determine the upper boundaries of the apices of the lungs; then, in the same way, the infraclavicular spaces are percussed. On the two sides in finger-percussion we must, if possible, hold the pleximeter hand in such way as always to have the wrist toward the middle line of the thorax and the pleximeter finger pointing outward.

Then we percuss the third intercostal space right and left, then downward only on the right, and usually only in the intercostal spaces. We do not further compare it with the left side, since here lies the heart, which is percussed by itself. Then follows the determination of the right lower border of the lungs according to the rules given above regarding the determination of parietal organs. We percuss upward, comparing the two sides of the thorax, again in the intercostal spaces. When we wish to percuss high in the axillæ the arms are to be abducted. Then follows the determination of the boundaries of the right and left borders of the lungs in the middle axillary lines. Sometimes it is valuable also to percuss from the infraclavicular spaces sideward and downward upon a line which is at right angles with the course of the ribs.

In percussing the back we first compare the sound over the apices of the lungs, thus completely defining their upper boundaries; then we percuss on the right and the left, comparing corresponding intercostal spaces as we proceed downward to the lower borders of the lungs. Then we percuss on the sides of the spine below the angles of the scapulæ, comparing symmetrical points. The boundaries of the lungs are best determined in the scapular lines.

In this way the thorax is generally to be percussed. But the presence of pathological conditions that require one to be especially careful in the examination of certain parts may give the preference to special methods of examination. These have been in part already mentioned in the general division. They follow directly from what was said there. They will be again mentioned in the description of percussion in pathological conditions of the lung.

[1] See below.

2. Normal Sound over the Lungs, Trachea, and Larynx.—The Normal Boundaries of the Lungs.—It is shown that in percussion of the lungs in general over the normal lung there is elicited a non-tympanitic sound. But this sound as regards its intensity is *individually very different* in different persons; also, in each single chest it is not alike throughout, but exhibits individual *regional differences.*

The individual variations arrange themselves first *according to the amount of fat.* Very fat bodies give a less clear thoracic sound, or in order to yield a clear sound they must be percussed more strongly, requiring perhaps the use of the hammer; but it is evident, as we have said, that this is unfavorable for determining the boundaries, for which the rule is to employ very light percussion.[1]

Further, the percussion-note of the chest differs *according to age:* with children, having a more elastic thorax, as well as with aged persons, with thin structural coverings and somewhat lax or rarefied lungs, it is higher in pitch than in persons in middle life.

But also in the individual thorax the different regions normally give different clearness of sound. In other words, one region compared with another yields a relatively deadened sound, and according to the two chief points of view previously mentioned—namely, according to the varying thickness of the covering and according to the size of the lungs. Hence we remark the following facts:

(*a*) Over the apices of the lungs, even with strong percussion, the sound is not very intense; for, though the covering is thin, the volume of lung-tissue is small.

(*b*) In the infraclavicular spaces, and still more in the second intercostal spaces, the sound is very intense (covering thin; volume of lung-tissue greater).

(*c*) *Farther down*, not only in the male, but in still higher degree in the female, the sound is deadened by the pectoral muscle or by this and the mamma; in the female the sound may be absolutely deadened over the mamma, and this notwithstanding the fact that the lung-tissue is here very considerable.

(*d*) *Upon the back* the apices yield a sound of very slight intensity, since here there is a very small volume of lung and a very thick body of muscle *Over the scapulæ* there is likewise a very deadened sound—at the spine and directly below even a thigh-sound. In the *interscapular spaces* the sound is clearer.

(*e*) *Below the scapulæ* and at the *sides of the chest* the sound is very intense.

(*f*) Strictly speaking, here also belongs the description of the so-called "*relative heart- and liver-dulness.*"[2]

Now, it is further very important to know which similarly situated points on the thorax normally give the same kind of sound, since it is especially by *comparative percussion* that we seek to ascertain the presence of disease on one side. We may say that in healthy people marked *dissimilarity of sound* at symmetrical parts of the chest on the right and left sides exists only—

In the neighborhood of the heart, as compared with the corresponding part on the right.

[1] See above.

[2] See p. 108.

At the two sides: on the left side normally the sound, almost as far back as the spine and forward in front at varying height as far sometimes as the fourth rib, is often clearer than on the right, and of somewhat tympanitic tone (combining with the sound of the stomach or colon).

In addition, there is a slight inequality sometimes *posteriorly over the apices.* In right-handed persons the sound on the right side at that location may sometimes be met with a little less clear, because the muscles from use are somewhat more developed. On the left side, in left-handed persons, the case is reversed.

Lastly, it is necessary to mention a point of greater importance—that over the *whole of the sternum* there is a clearer, non-tympanitic sound even where there is no lung-tissue at all, as at the upper part of the manubrium (trachea) and over the left half of the lower part of the corpus sterni. The sternum acts as an unusually thick pleximeter, and yields, therefore, throughout and in equal strength, the sound of the lung lying in contact spread out over its inner surface.

The *larynx* and *trachea* in the neck in front give the tympanitic sound of a hollow cavity with smooth walls. This has the peculiarity of being higher and more plainly tympanitic with the mouth open than with it closed (*Williams's tracheal tone*, tracheal change of sound). The cause of this phenomenon is not quite clear; the explanation given by Neukirch and accepted by Weil is based upon the assumption of the resonance of the mouth changing with its opening and closing. This will be referred to later.

Normal Percussion-boundaries of the Lungs.[1]—It is not possible to define the boundaries of the lungs perfectly by percussion. Moreover, by percussion we can only establish—

1. The *apices* so far as they rise above the clavicle: they are distinguished by their clear sound in comparison with the deadened sound of their surrounding soft parts.

2. The boundaries of the left lung at the *incisura cardiaca:* the lung sound from the absolutely deadened sound of the heart—*the lung-heart boundary.*

3. *The lower borders of the lungs*, this especially at the lower border of the right lung: the lung sound marks the boundary of the absolutely deadened sound of the liver—the *lung-liver boundary.*

At the lower border of the left lung, first about from the mammillary to the middle of the middle axillary line, the lung sound marks the boundary of the tympanitic sound (stomach, or more rarely also intestines)—*lung-stomach boundary;* next, the lung sound from the deadened sound of the spleen—*lung-spleen boundary;* and, lastly, from the deadened sound of the kidney—the *lung-kidney boundary.*

It is difficult to determine the boundaries of the lungs, since the difference of sound is often slight, especially as the tympanitic sound of the stomach often mingles with the lung-sound higher up than the anatomical border of the lower limits of the lungs; moreover, the lower boundaries of the lungs close up to the spine on both sides, because of the thick layers of the erector spinæ, require strong percussion, and this is unfavorable for determining the boundaries.[2]

[1] Compare Figs. 31 and 32.

[2] See above.

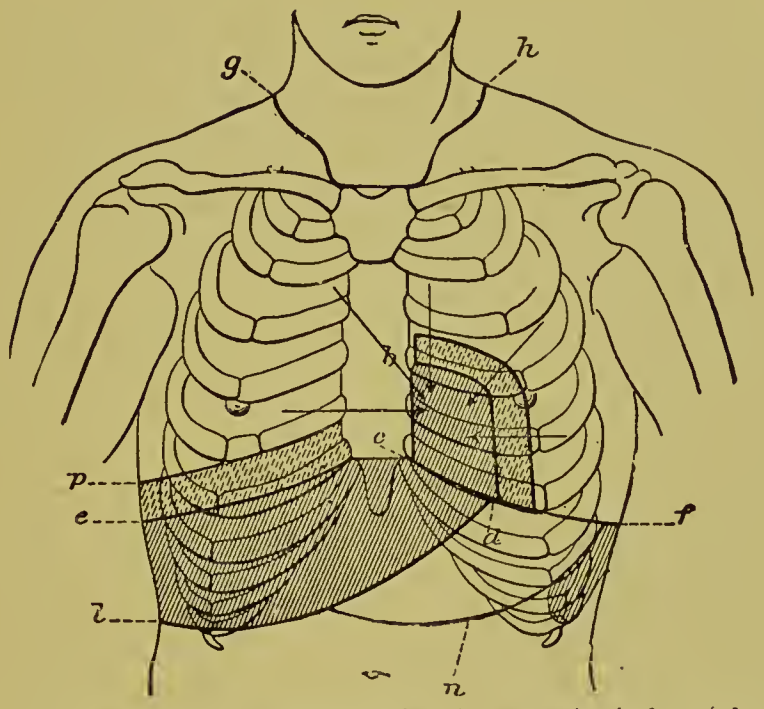


FIG. 31.—Boundary of the lungs as determined by percussion in front (after Weil).

g, *h*, the extent of the lung upward; *e*, *f*, the lower limit of the lungs; *b*, *d*, the relations of the lung and heart at the incisura cardiaca. The strongly-hatched surface represents the portions of the heart and liver which are parietal; the lighter hatching shows the so-called relative heart- and liver-deadness.[1]

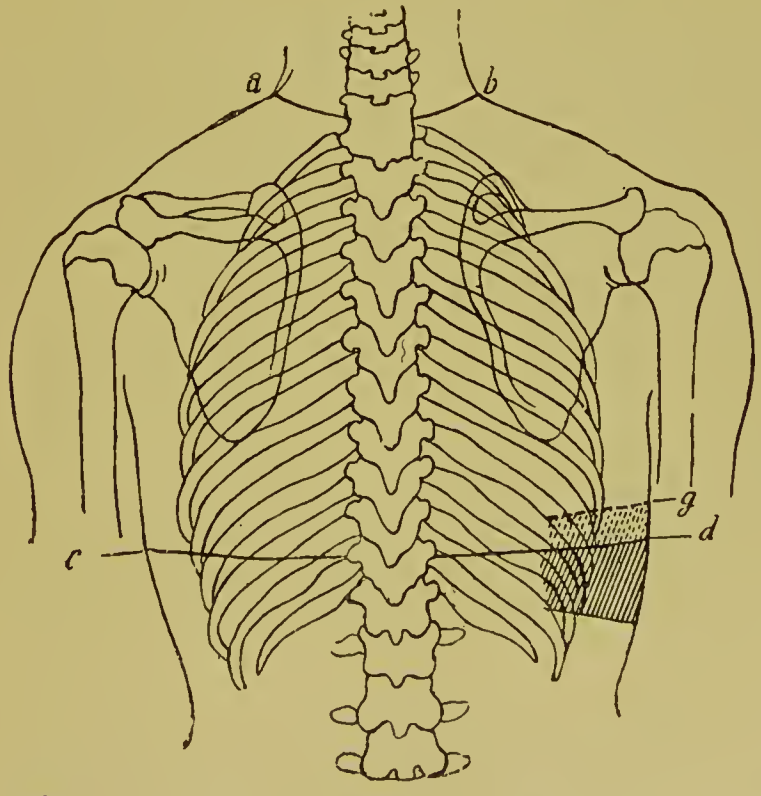


FIG. 32.—Boundary of the lungs as determined by percussion upon the back (after Weil).

a, *b*, the upper limits of the lungs; *c*, *d*, lower limits.

[1] See below.

We cannot determine by percussion the front borders of the lungs behind the sternum. This is the case because there the lungs lie close to each other for some distance, and also because the sternum, like a firm bone, yields a uniform sound, and it is not possible to recognize a difference of sound in what lies beneath it: it yields throughout a clear sound, very like the lung resonance over the ribs.

Hence, it may also be explained that the lower part of the anterior border of the right lung, which behind the sternum is limited by the heart, cannot be defined by percussion: we much more receive, instead of the actual boundary of the right lung, one that is apparent—where the uniform sternal sound is exchanged for the absolutely deadened sound of the heart at the left border of the sternum. In front the base of the right lung does not extend so far down as the left, the right coming as low as the inferior border of the fifth rib, while the left corresponds with the superior border of the sixth rib.

Relative heart- and liver-dulness. The determination of the lung-heart and the lung-liver boundaries is made more difficult by the peculiar circumstance that, on account of the small volume of lung-tissue at the border of the lungs, the resonance of the lungs immediately over the borders has very slight intensity, a relatively deadened sound. We percuss from the lung toward the liver with strong or moderately strong strokes, and find, say in the mammillary line at the fifth rib, a strong relatively deadened sound which the beginner is inclined to regard as absolute liver-dulness. But this, as has been said, corresponds with the thinning of the lungs at the lower border. In this way a zone of relative dulness manifests itself over the whole of the lower border of the right lung, except close to the spine behind, and in a similar, but somewhat smaller, zone the heart-dulness bows round and to the left: this is the (incorrectly) so-called relative liver- and relative heart-dulness, as indicated by the light shading in Figs. 31 and 32. Also, sometimes, there is such a relative dulness over the lung-spleen boundary. It does not exist over the lung-stomach boundary, because here, by moderate percussion, the coincident sound of the stomach causes a low tympanitic sound.

These zones are diagnostically important only in isolated cases, and they have nothing to do with enlargement of the heart, liver, or spleen.

In order to avoid deception by these conditions when determining the boundaries it is necessary to take care—

1. To percuss lightly in determining the boundaries of the lungs.

2. To mark the lung-heart and the lung-liver boundary—that is, the border of the lungs where the relative dulness passes into absolute dulness, or, in other words, where, in percussing from the lungs toward the heart and the liver, the dulness begins to be so marked that it no longer increases.

On the average—that is, in middle life—we thus find (compare Figs. 31 and 32) *the lung-liver boundary*, in the mammillary line at the sixth, in the middle axillary line at the eighth, in the scapulary line at the tenth rib; *the lower border of the left lung*, in general as high as the right only in the mammillary line at the lower border of the sixth rib; *the lung-heart boundary*, at the fourth rib and more or less just without

the parasternal line; *the upper limits of the apices of the lungs*, three to five cm. above the clavicle.

Differences by reason of age. In children the lower border of the lungs is from a half to a whole intercostal space higher; in old persons it is that much lower (Weil). There is a like difference as regards the lung-heart boundary. That is, the lungs increase with the years as compared with other organs.

Displacement of lower border of the lungs is manifest by percussion:

1. In deep inspiration and expiration (active mobility): in the middle axillary line the lower border sinks with deepest inspiration about three to four cm.; in the mammillary and scapular lines, about two to three cm.; in deepest expiration it rises up not quite so much above the average location (Weil). With deep inspiration at the incisura cardiaca the lung moves so as quite to cover the heart, and it may even entirely obscure the heart-dulness.

2. In change of position (passive mobility): when lying upon one or the other side the lower border of the lung of the opposite side moves down as much as three to four cm. (Gerhardt, Salter, Weil).

3. Abnormal Sound over the Lungs. Abnormal Position of the Border of the Lungs.—A. Dulness: Deadened Resonance.—In order not to overlook slight deadening we must remember what was said upon comparative percussion on pages 104, 105; if the comparison with the opposite side is inadmissible, as when both sides are diseased, then the comparison is made with the adjacent parts upon the same side, bearing in mind the normal regional differences of intensity of sound.[1]

Thus, in *disease of both apices* we sometimes recognize the deadness of the apex to be less affected by comparing the resonance over the latter with the percussion-resonance a little lower down, remembering that normally the resonance over the first and second intercostal spaces must be clearer than in the supraclavicular space, and clearer than over the third intercostal space.

But also, without further consideration, we must not designate every deadness as due to an internal organ, but consider the deadening influence of a sharply-bowed rib, etc. *Slight deadening*, without any other pathological evidence, especially over the apices, is to be given value with very great caution.

Resonance is deadened:

(*a*) *By the development of airless tissue in the lungs, either by condensation or by solid new formations in them.*

In *croupous pneumonia* the lung-tissue in the height of the disease is in the stage of hepatization. Generally, in a large region it is completely deprived of air through the filling of the alveoli with inflammatory exudate. An *intense deadening* is coextensive with this condition. It seldom becomes absolutely deadened like the thigh-sound, but there can generally be recognized a slight tympanitic tone. The *feeling of resistance* is generally likewise correspondingly increased, but not so much as is the case with a pleuritic exudate.

Thigh-dulness and very marked feeling of resistance may exist with *croupous pneumonia* if, besides the lung-tissue, the bronchial tubes of

[1] See above.

that part of the lung are likewise completely filled with the exudate ("massive pneumonia"), or if the croupous pneumonia is complicated with a large pleuritic exudate, which is then almost always behind and low in the chest. The extent of the deadening in croupous pneumonia very frequently corresponds with a lobe of the lung, because of its being a lobar pneumonia, or there is evidence of an enlargement of the lobe in all directions (the inflammatory exudate spreads out to a considerable extent). Often, therefore, in this disease we may recognize the boundaries of the lobe in the figure of the area of deadening, or the boundaries which correspond to the tracing of the lobe enlarged in all directions. The infiltrated part of the lung may, however, be also smaller, especially on the surface of the lungs, occupying so small an extent as not to cause any recognizable deadening. Auscultation[1] here leads to a conclusion sooner than percussion.

In the neighborhood of an infiltration the resonance is generally abnormally loud and deep, even slightly tympanitic (compare what is said of croupous pneumonia under B. Tympanitic Sound).[2]

Since the infiltrated lobe of the lung is somewhat larger than normal, sometimes in pneumonia of the whole lower lobe deadness will be found posteriorly as far up as the apex without the apex being involved. On the contrary, percussion upon the front of the chest, over the apices of the lungs, then yields a clear and, in consequence of the relaxation, a very loud, deep sound. Further, for the same reason, in pneumonia of the left lower lobe the lower border of the deadness may overstep the region of the normal boundaries of the lungs, as the marking out of the lung-stomach boundary then shows that the so-called "halfmoon-shaped space" is somewhat smaller.[3]

Also in *catarrhal* or *lobular pneumonia* and tuberculosis (in the so-called infiltrated tuberculosis of a larger part of the lungs) there may be an extended thickening and a corresponding deadening. Often, indeed, there are pathological deposits so small that their presence is not revealed by percussion, but, though widely scattered, they are interspersed with points still containing air, and hence give a clear sound. Then, because the tissue of the parts still remaining normal is somewhat lax, the resonance is often tympanitic, or the latter sound is mingled with that of deadness from the infiltrated parts—the tympanitic deadened sound.

In *tuberculosis of the apices* of the lungs there is, at the beginning, in very slight measure, a mingling of thickened parts with tissue containing air, but relaxed; hence the resonance in the beginning over the diseased apex is very often tympanitic or tympanitic-deadened, in comparison with the healthy apex. Moreover, there is early retraction of the upper boundary of the apex upon the affected side.[4]

Large hemorrhagic infarctions and sections of the lungs compressed even to the point of not containing any air, as from pleuritic exudations, tumors, and large pericardial exudations, may likewise give a deadened sound. Finally, it is conceivable that solid tumors of the lungs (sarcoma, carcinoma) produce the same effects if they lie upon the surface or attain to a certain size.

[1] See Auscultation. [2] P. 112. [3] See under Digestive Apparatus.
[4] See under Diminution of the Boundaries of the Lungs.

(*b*) *Resonance is deadened by the presence of a deadening medium over the lungs*—that is, between it and the percussing finger.

Most important of these is *pleuritic exudate*. Generally, this first appears low down posteriorly in the complementary space and above it, and if it amounts to as much as 400 cubic cm., it may even be recognized by light percussion. Corresponding with the increase of the exudate the area of deadness will gradually become more extensive; its limits ordinarily correspond with a fluid surface which, while the patient is in the posture most frequently assumed, is somewhat horizontal; that is to say, in bedridden patients the fluid levels itself high up on the posterior wall of the thorax, and the limits on the sides and in front drop off sharply, while with people who are much out of bed or may still be at work the fluid stands equally high in front and at the back of the chest. When the effusion is very large the deadness may extend even to the apex, both anteriorly and posteriorly. When the effusion is considerable, it quickly causes an absolute deadening and with the most marked feeling of resistance.

Corresponding with the increase of the fluid the lung becomes lax in an ever-increasing area, since it may then follow its elastic traction; immediately over the fluid it gives deadness, and when there is a large exudate, where at least there is ordinarily left a district with clear sound—namely, high in front—it yields an abnormally loud and deep, or a tympanitic sound, sometimes *cracked-pot sound*.[1] A very large exudate may even compress the lung to such a degree as to expel all air.[2]

When there is a certain amount of exudation its weight presses upon the diaphragm, increases the affected pleural cavity toward the side, presses out the side of the thorax,[3] and pushes the mediastinum and the heart over toward the sound side.[4] The downward pressure of the diaphragm in cases of pleurisy of the right side is recognized by the liver being lower.[5] In pleurisy of the left side it may be made out directly by locating the upper boundary of the so-called "half-moon-shaped space."

When the pleural surfaces directly over the exudate are glued together, then in change of position of the patient the pleuritic exudate is not movable, and the boundaries of the deadness are therefore not changeable; not infrequently the exudate is entirely "capsulated" by the adhesion of the pleural surfaces. If the exudate is reabsorbed, then the evidences of expansion and of displacement, on the one hand, and the deadness (and, indeed, according to its extent, likewise its intensity), on the other hand, steadily disappear. Often the upper border of deadness then shows as a bowed line with its convexity upward (*Damoiseau's curve*).

If a new pleuritic exudation takes place between pleural surfaces already adherent from a former attack, then, of course, it remains confined within the space thus prepared—"encapsulated, circumscribed pleurisy." The boundaries of the exudate may, in these cases, take a very varying course.

Hydrothorax practically gives rise to similar appearances, but it is

[1] See page 116. [2] See above. [3] See above.
[4] See Displacement of the Heart. [5] See Percussion of the Liver.

generally on both sides, yet not infrequently with a very different amount upon the two sides. Further, hydrothorax always shows in change of position, although only after a certain time, a change of its relation to the thorax in such a way that it tends to take possession of the part of the thorax that, for the time being, is the lowest; accordingly, there is what may be called a passive mobility of the boundaries of deadness.

Serous or purulent or ichorous effusion into the pleural cavity complicating pneumothorax (sero-, pyopneumothorax) is distinguished from the above by its mobility with the change of posture. It behaves like water in a bottle when the position of the latter is changed; in every situation the fluid maintains a horizontal surface, and occasions at the same time, with every change of place or location of the thorax, a prompt variation of the upper boundaries of the deadness.

Further, a deadening of the resonance is occasioned by the *thickening of the pleura*, which either remains after an exudative pleuritis or in conjunction with processes slowly going on in the lungs. The latter is very frequently the case in tuberculosis of the apices of the lungs: marked deadening, appearing early in the beginning of the disease, is generally caused by pleural thickening. The intensity of the deadness is determined by the amount of the thickening; it may even become like thigh-deadness. The feeling of resistance is generally very markedly increased; with very thick deposit this is positive. Tumors, as a matter of course, likewise cause deadening. This latter deadening generally exhibits an irregular boundary, unless, as is rarely the case, complicated by pleuritic exudations.

It is sometimes very difficult to distinguish between a thickened pleural surface and a portion of pleural exudate left behind with moderate thickening; this question often arises especially where the deadness is low down posteriorly. In arriving at a decision the first thing to consider is whether there is expansion or contraction, or whether there is a deep or a high position, of the diaphragm.

But here, as well as in the often very difficult differential diagnosis between pleural exudations and tumors, as of the lungs, pleura, or chest-wall, the application of the explorative puncture is the best means of deciding.

Finally, the resonance of the thorax is deadened by all processes in the chest-wall which lead to its being thickened—tumors, peripleuritis, edema.

The second quality of sound which is found over diseased lungs is—

B. Tympanitic Sound.—It occurs pathologically: (*a*) if the lung is in a state of *elastic equilibrium*. We know that this condition is a consequence of retraction of the lung, with large pleuritic exudation as well as shrinking in connection with pleurisy; further, in all other affections of the chest which decrease its capacity. Hence tympanitic resonance exists over the lungs in the neighborhood of tumors of all kinds; sometimes in the neighborhood of the heart in exudative pericarditis, more rarely in hypertrophy and dilatation of the heart; lower in the thorax: in diaphragmatic pleurisy; in high position of the diaphragm from subphrenic tumors, abscesses, etc.; and in general peritonitis from general distention of the abdomen by ascites, tumors, etc.

We may also think of the same condition of approaching equilibrium of elasticity as arising from *relaxation* of the lung-tissue (Weil); and this will explain the tympanitic resonance that exists with *croupous pneumonia* in the stage of engorgement and resolution; over many small *catarrhal-pneumonic* and *tubercular deposits*, since the intervening tissue containing air has become lax; and, finally, in *edema of the lungs*.

(*b*) In consequence of *marked shrinking and thickening of the lung*, in strong percussion of the supraclavicular fossa, it arises from the trachea, while in percussing the first or second intercostal space it arises from this or the primary bronchus, directly from the percussion-blow, and so the broncho-tracheal column of air is put in vibration; there is thus produced a peculiar change of sound in the trachea, the sound with the mouth open being more distinctly tympanitic and higher (*Williams's tracheal sound*).

(*c*) *Over cavities within the lungs, caverns* (*vomicæ*).

We may have here, according as the cavity does or does not communicate with the outer air by means of a pervious bronchial tube, the *open* or the *closed tympanitic resonance*. In the former case the sound under all circumstances is more distinctly tympanitic, and also more intense; in the latter case, on the other hand, it is much less distinct and weaker, all the more since we must assume that the cavities, because they lie in the thorax, have more or less stiff walls, and since the rigidity of the wall with the cavity closed hinders the condition that causes the tympanitic sound.[1]

How large the cavity must be in order to give a tympanitic sound it is not possible exactly to state, since, besides the size, the situation of the cavity (whether parietal or deep), the amount of fluid secretion it contains, its walls (whether smooth and vibratory), the condition of the surrounding lung-tissue, and finally the vibratory capacity of the given thorax, must also be taken into consideration. Generally, cavities occurring in the apices from tuberculosis exhibit more distinct physical characteristics than cavities in the lower portions of the lungs, which frequently are of the nature of bronchiectasis, since the former, even when of moderate size, must reach to the surface of the lungs, and generally have thickened walls. Cavities as large as a walnut in the upper parts of the lungs generally give a distinctly tympanitic resonance.

If the cavity is very large with relatively smooth walls, a metallic tone is added to the tympanitic resonance.

If the cavity is covered by thickened lung-tissue or with thickened pleura, as is frequently the case, then the sound becomes *tympanitic-deadened;* if covered by a very thick layer of airless tissue, possibly absolutely deadened.

Temporarily marked filling of the cavity with secretion deadens the tympanitic sound also, sometimes even to absolute deadening; further, the sound becomes temporarily indistinctly tympanitic and deadened-tympanitic if a bronchus connecting with it, otherwise open, becomes closed (with secretion or from dipping below the fluid contents of the cavity).

[1] See p. 97.

Under different conditions *a tympanitic sound* over a cavity *may change its pitch:*

1. The so-called *simple Wintrich's Change of Sound.* The tympanitic sound becomes louder, more distinctly tympanitic, and higher if the patient opens the mouth wide and, as is desirable, at the same time protrudes the tongue a little. This can only occur over those cavities that freely communicate with the broncho-tracheal column of air.

We percuss, not too strongly, while the patient lies or stands quietly and alternately opens and closes the mouth; but it is necessary for the patient to breathe as superficially as possible, or we must compare the sound in the same stage of the breathing, since the sound also sometimes changes its pitch according to the stage of the breath.[1]

The explanation of this symptom, as of the tracheal change of sound, which is exactly similar, is that it is from the change of consonance of the mouth-throat cavity. [See preceding page.—Tr.]

This *Wintrich's change of sound* may also occur over cavities in such a way that the sound with the mouth closed is markedly deadened, with only a trace of tympanitic sound (especially with marked callous formations over the cavity), and only with the mouth open does the sound become tympanitic (at the same time becoming louder and noticeably higher).

I would like, therefore, in opposition to Weil, to insist that we ought, if there is only a slight possibility of the existence of a cavity, and also in the case of tympanitic sound slightly distinct, or indistinct, with dulness, to apply the test of Wintrich's change of sound.

It is very easy to confound the *simple Wintrich's change of sound* with *Williams's tracheal sound.* We should take notice—(1) Whether there is very marked contraction, when it is much more likely to indicate change of sound than Williams's tracheal sound; (2) Whether in order to cause the change of sound only weak percussion (cavity) or strong percussion (trachea or bronchus) is required; (3) Whether there are other symptoms of a cavity.

Simple Wintrich's change of sound points with greater probability to a cavity. But its value as an indication is diminished by the above-mentioned possibility of being confounded with Williams's tracheal sound.

2. *Interrupted Wintrich's Change of Sound* (Gerhardt, Moritz). It is distinguished from the simple in that in some positions of the body it is plain, in others it is indistinct or is wanting. The explanation of this is that in one position the bronchus leading to the cavity is open, while in the other it dips into the secretion in the cavity, and so is closed. In this way the tracheal change of sound cannot possibly be interrupted.

This change of sound is very rarely met with, but it is to be regarded as a positive sign of a cavity.

Rumpf[2] recently found in four patients Wintrich's change of sound, which only was manifest if the acme of sound was always compared with the acme of inspiration; which change, however, was always

[1] Compare under 4, Respiratory Change of Sound, p. 115.

[2] *Berliner klin. Wochenschrift*, 1890.

absent during other phases of respiration. At the autopsy of all four cases cavities were found. Rumpf quite plausibly draws the deduction from obvious physical reasons (which we cannot here follow) that such a change of sound must necessarily accompany a pathological cavity. Parenthetically, we remark that, according to Rumpf's description, it seems to us that what he observed is analogous to Seitz's metamorphosing breathing.[1] We must wait for further observations.

The designation "respiratory change of acme of sound" we consider to be questionable, for the reason that it may cause confusion with Friedreich's respiratory change of sound.[2]

3. *Gerhardt's Change of Sound.*—A tympanitic sound changes its pitch if the patient changes his posture (upright, dorsal, side position); and sometimes, if the patient changes from the dorsal to the upright position, the sound becomes deadened-tympanitic or absolutely deadened over the lower part

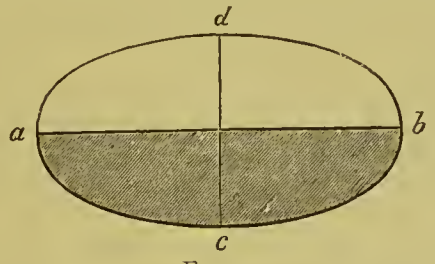


FIG. 33.

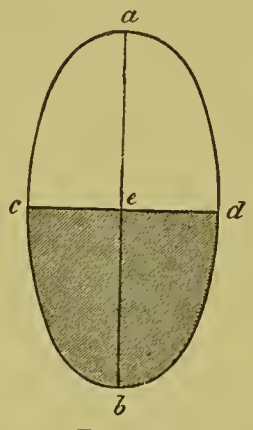


FIG. 34.

FIGS. 33, 34.—Gerhardt's change of sound. Schematic representation of the behavior of the contents of a cavity with a change of position of the body of the patient.

of the cavity, because in this position the fluid contents of the cavity come into contact with the chest-wall.

Gerhardt's change of sound may take place over communicating, as well as over closed, cavities. The change of pitch, in case the cavity is open, may have very different causes, which we will not discuss here. In closed cavities it is really due to a change in the tension of the chest (and cavity?) wall, perhaps also to a change in the size of the part of the cavity containing air—a change caused by different location of the secretion. (See Figs. 33 and 34, from *Weil's Handbook.*)

Gerhardt's change of sound is in every form an almost certain symptom of a cavity, but, like the preceding, it is very rare.

4. *Friedreich's Respiratory Change of Sound:* the sound becomes higher at the height of a deep inspiration. This occurs not alone over cavities, but may be observed in any case of tympanitic sound over the lungs. It depends upon the increased tension during inspiration of the chest-wall and lung-tissues, likewise of the wall of the cavity.

It does not have diagnostic significance. But it is important to know it in order that we may not be misled by it in the examination of other changes of sound; therefore, we ought in testing this to observe the rule to percuss during very superficial breathing, or, still better, always to percuss at the same stage of the breathing, as has been said above.

[1] See p. 127.

[2] See below.

(*d*) Finally, the *tympanitic sound* occurs in very rare cases in pneumothorax, and sometimes entirely in cases that have permanent and completely-open fistulæ; this "open" pneumothorax is generally circumscribed. In pneumothorax the tympanitic sound may sometimes exhibit Wintrich's change, since the physical conditions upon which it depends are also present, as in large communicating cavities: open communication of an air-space with the broncho-tracheal column of air. Here we have also metallic tone (see p. 113).

Cracked-pot Sound (*Bruit de pot fêlé*).[1]—It seems that this is the most suitable place to describe this phenomenon, which, while very surprising and remarkable, is of very subordinate diagnostic significance. It consists of a peculiar click ("clinking of coin" or "trembling") which sometimes accompanies the clear sound, and indeed generally the tympanitic, more rarely the clear, non-tympanitic. It corresponds to the noise which occurs when we strike a cracked plate or pot, or when we hold the palms of the hands together lightly and then strike them upon the knee. It occurs in the thorax if an instantaneous current of air of a given force is driven by the percussion-stroke from the lungs against the larynx, or if during expiration a stream of air flowing outward is for a moment suddenly sharply arrested. This symptom requires strong percussion, yielding thorax, and thin covering (generally in front superiorly and also below posteriorly). It sometimes occurs in normal cases, almost only in children.

Pathologically it occurs—

1. Over *large parietal cavities*, here often remarkably intense.
2. In *pneumothorax with open fistula*, especially if circumscribed.
3. Over *pneumonic deposits*.
4. Over *retracted lung-tissue*, especially above large pleuritic exudates (high in front), rarely in the neighborhood of thickened portions of lung.

This phenomenon is always more distinct if we percuss during expiration; very often, especially in case of cavity and open pneumothorax, it becomes louder by opening the mouth.

As above remarked, this symptom has almost no diagnostic meaning, since it is present with such varying conditions.

The noise is caused by a swift current of air striking at a narrowed point: this happens at the glottis, in a cavity at the mouth of a bronchus, and at the puncture in the pleura in case of pneumothorax. Sometimes a rattling sound is mingled with the trembling ("the moist cracked-pot sound").

C. Abnormally Loud and Deep Sound.—This occurs—

1. In *severe emphysema of the lungs*, designated as "*band-box note*" (*Biermer*).

2. In *decreased tension of lung-tissue* above a pleuritic exudate. A zone of this abnormal sound lies just above the line of deadness produced by an exudation in the neighborhood of pneumonic thickening —as anteriorly in pneumonia of a whole lower lobe; sometimes in the neighborhood of the heart in pericarditis exudativa, but also with dilatation and hypertrophy; likewise and especially, in the neighborhood

[1] Referred to on p. 111.

of encroaching tumors, and with a high position of the diaphragm consequent upon abdominal affections.

As was said before, in most of these cases, if the tension of the lung-tissue is very considerable tympanitic resonance may arise.[1]

3. *With Pneumothorax.*—Here the sound, in consequence of the strong tension of the chest-wall, is almost always non-tympanitic, loud, and deep. Only (a rare case) in open pneumothorax, especially if it be circumscribed, is tympanitic sound sometimes met with.[2]

This abnormally loud and deep, even tympanitic sound of pneumothorax gives almost regularly the *metallic sound*,[3] only seldom recognizable, however, by the ordinary methods of percussion, but very admirably by the *rod-pleximeter percussion* described by Heubner.

Mode of Application.—Rod-pleximeter percussion is best conducted by two examiners. One strikes with the handle of the percussion-hammer or with a pencil upon a pleximeter; the other auscults the thorax. If both manipulate over a pneumothoracic cavity, the second hears the strokes as the finest metallic, generally a silvery, clear ringing.

This, moreover, is sometimes also observed with very *large* and *smooth-walled cavities* with thin covering. With pneumothorax accompanied with fluid (pyo-, seropneumothorax) the metallic sound, almost without exception, changes its pitch with the change of position; in sitting up it is generally deeper, but sometimes also higher (*Biermer's change of sound*). If the effusion is so large as entirely, or almost entirely, to fill the pleural cavity, of course the metallic sound disappears.

It will be mentioned in the appropriate sections that this metallic ringing in pneumothorax not only accompanies such an artificially created sound, but also may be present with rhonchus, respiratory sound, and heart-sound.

D. Changed Condition (and Diminished Power of Displacement) of the Boundaries of the Lungs.—(*a*) *Extension of the boundaries of the lungs* takes place in *emphysema:* the lower borders usually move sidewise and deeper, in the most marked cases, both front and back. The mammillary line will be at the eighth rib, the axillary line at the tenth, the scapular line at the eleventh or twelfth. Heart-deadness may almost or quite disappear from the expanded lung lying over it from the side. At the apices of the lungs sometimes a slight enlargement of the lungs may be made out; in rare cases even expansion of the apices may likewise take place (as after whooping-cough in children). "Relative liver-" and "heart-dulness" is very small; simultaneously with the expansion the lung loses its power of displacement, both active and passive, even past recognition.

One-sided downward movement of the boundary of the lung occurs in vicarious emphysema, but the capacity to change its boundaries is preserved in this case.

Apparent one-sided expansion of the boundary—that is to say, *the appearance of a clear sound upon one side quite beyond the normal boundary of the lung*—takes place in diffuse *pneumothorax:* the lower border of the clear sound is sometimes met with even deeper than in

[1] See p. 112. [2] See p. 116. [3] See pp. 113, 116.

emphysema. This border is immovable, and always very sharply defined. The side of the thorax is expanded, the heart and also the liver are displaced, or the tympanitic sound of the "halfmoon-shaped"[1] space is replaced by the sound of pneumothorax. Displacement of the mediastinum in right-sided pneumothorax is generally distinctly recognized by the change of sound between it and the left lung (the boundary-line lies to the left of the upper part of the sternum).

(*b*) *Diminished volume of the lungs* is shown by the lower boundaries of the lungs being higher than normal on both sides, by the diaphragm being pressed up from below, or from its being paralyzed; one-sided diminution, by shrinking from disease of the lung or a past pleurisy. The motility of the borders is thus diminished or destroyed. The liver stands correspondingly higher[2] or the "halfmoon-shaped" space is enlarged.

Sometimes in phthisis diminution in size of an apex manifests itself by the deeper position of the upper border of the lung upon one side.

(*c*) *Diminution of the motility alone*, especially during respiration, without change of the average condition of the borders, sometimes exists low down posteriorly as the *first symptom of pleurisy*, and also as the only sign of a past pleurisy, in which case it is noticed along the whole lower border of a lung or a part of the same, as at the heart; here, also, it is a residuum of pericarditis externa.[3]

Retraction of the lungs in the neighborhood of the heart by shrinking permits the latter to come in contact with the chest-wall to a larger extent than normal; there is displacement of the heart-border of the lung to the left and upward, and hence hypertrophy or dilatation of the heart may at first be mistaken for the real condition.[4]

On the other hand, diseased conditions in the neck (tumors, scars, etc.) may influence the position of the apices, and thus at first may deceive the inexperienced examiner in leading him to conclude that there is one-sided shrinking of the lung.

Auscultation of the Lungs.

1. History.—The Sphere of Auscultation at the Present Time.—It now appears to us very strange that the idea of percussing the body was only so lately brought into medical practice. It is yet more difficult to understand that *methodical auscultation of the body* is only a child of the most recent time. It is true that Hippocrates heard what he named a succussion-sound, and also, no doubt, rattling and rubbing sounds, but to the latter two he did not attach any great importance; and in all the centuries from the Greek physician to the time of Laënnec no real attention was given to the audible phenomena of the healthy or diseased body. Only a few voices—that of the often-mentioned Hooke more than any other (second half of the seventeenth century)—were timidly raised, and these were not heeded. Only in consequence of the discovery and general consideration of the value of percussion was auscultation developed, and this by Laënnec, the discoverer of the stethoscope. His epoch-making work is called

[1] See under Percussion of Stomach.
[2] See Percussion of Liver.
[3] See Examination of the Heart.
[4] See Examination of the Heart.

Traité de l'Auscultation médiate et des Maladies des Poumons et du Cœur. After him, Skoda, by critical sifting and by his own efforts, which traced the new phenomena to their physical causes, rendered imperishable service to this branch of knowledge. But up to the present time the work has still been going on, which, in part, has made new discoveries, and, in part, has investigated the nature of what was already known.

The sphere of auscultation—of listening—in its widest sense extends to all that we are able to take note of by the ear, hence, in the first place, to the voice, cough, sounds caused by breathing, by mucus in the upper air-passages, which may often be heard in the farthest corner of the sick-chamber. But, strictly speaking, auscultation concerns only those phenomena which the ear perceives, either by direct application to the body or which are brought to it by an instrument, as a stethoscope or an ear-trumpet. These, so far as they refer to the respiratory apparatus, form the subject of the following section.

2. Methods of Auscultation.—Nowadays we employ both the immediate (direct) and the mediate (indirect) auscultation. In the first the ear is directly applied to the person to be examined; in the latter we employ a stethoscope or ear-trumpet. While, as will be referred to later, we employ almost exclusively the indirect method in examining the heart and vessels, both methods are applied in the examination of the respiratory apparatus, and particularly of the lungs. In applying both, where it is possible we must endeavor to have the body bare; in no case should the covering be more than a single thickness, and that should be as thin as possible and must be perfectly smooth.

The *application of the ear* to the body consists simply in laying the ear lightly over the particular part to be examined. In order to place the ear exactly over the spot which we wish to auscult, it is well to place the tip of the index finger at the point and keep it there until the ear is placed at the point indicated, when the finger is withdrawn. For *stethoscopic auscultation*, in Germany at the present time, preference is almost universally given to the simple hollow stethoscope, the tube being about twelve to eighteen cm. long, with a not too small ear-plate. No doubt the plate has this disadvantage—unless the examiner is sufficiently careful—that it does not lie smoothly upon the outer ear; but, nevertheless, it is the most suitable form, since the stethoscope with hollowed ear-pieces, especially those recently devised, which, embracing the head of the auscultator, lie over the whole outer ear, for most persons have a most disturbing roar—a disadvantage which quite outweighs the advantage that, by increasing the resonance, it so well conducts the noises from the body; and the cone-shaped ear-pieces which are inserted into the outer ear in the short stethoscopes with stiff tubes cannot long be borne by the examiner. These short stethoscopes may have the further peculiarity that the end that rests upon the body measures, on the average, not more than two to five cm., hence they conduct to the ear impressions of sound from a much smaller region than will be heard from by direct auscultation. They are made of various materials (wood, hard rubber, ivory), but this is of small importance. The *flexible stethoscopes* (rubber tubing instead of the stiff tube, and ear-cones instead of the ear-plate) come less into

use because it is difficult, at least in the beginning of their use, to exclude the marked noises that are associated with them. Of the *double stethoscopes* I only mention that of Camman. Though somewhat complicated, it is very useful when one has learned to disregard the accessory sounds, which it intensifies as well as those one wishes to listen to.

In general, the use of the stethoscope resembles the practice of percussion in that every one, especially while learning, ought always to use the same kind of instrument, in order that they may learn to judge correctly of the auditory impressions which the instrument furnishes. In my teaching I have always found that those students who each time they wish to make an examination had to borrow an instrument from their fellows did not hear anything.

There are a large number of forms of stethoscopes, especially of the hollow stiff ones, the separate models of which it is not possible to describe. It may be remarked that the microphone has recently been employed.

[By the use of a solid—a wooden or hard-rubber—stethoscope, referred to below, it is not absolutely necessary to remove the clothing; by pressing the instrument *firmly* against the chest with the fingers friction of the clothing is prevented.

The phonendoscope, devised by Bianchi of Florence with the assistance of Bozzi,[1] marks a great advance in the means of auscultation. Its field of application is rapidly widening. By its aid minute changes are readily made out which before its use were not suspected. The contour of organs can be distinctly mapped out.—*Translator.*]

P. Niemeyer's solid stethoscope with ear-cones (acuoxylon) is decidedly not to be recommended; it has not proved practical, nor are the theoretical grounds of its construction sound.

It is very important in the beginning not to make pressure with the stethoscope. This must be considered particularly if, as it is often desirable to do, one removes his fingers from the stethoscope after having applied it to the desired place, and then holds it in place by means of the ear. However, in order to distribute the slight unavoidable pressure a rubber ring may be used, which is drawn over the foot of the stethoscope. The ring must be frequently renewed. But with proper care it may be entirely dispensed with.

As was said above, it is decidedly to be recommended in the examination of the lungs to employ both direct and indirect auscultation. The former is here preferable, since by it we can generally listen at one time to a large region of the lung; hence it is, on the one hand, more comprehensive, and, on the other hand, furnishes collectively louder sounds. Moreover, in the examination of the chest posteriorly of very sick patients it cannot be dispensed with, since by its comprehensiveness it furnishes the means of conducting the examination with the necessary quickness. On the other hand, the stethoscope is employed—

1. Where the ear cannot be applied, as over the supraclavicular spaces.

2. If we wish to listen quite separately to noises existing in a narrow, limited space.

[1] *Medical Record*, N. Y., Oct. 31, 1896, p. 624 f.

3. Sometimes, from reasons of delicacy, as over the female breast.

4. If the physician wishes to avoid being soiled, or the risk of receiving or getting parasitic insects or infections.

In a general examination it is well to auscultate after percussing. After percussing the front of the chest, auscultate over the same region, and then percuss and auscult the back. Generally the patient should breathe deeply; it is not at ali preferable to have him breathe very hard and quickly. Not infrequently we hear best with moderately deep breathing. Where it is possible, as in percussion, symmetrical parts should be compared. The particular points where it is necessary to take care are described in the following section.

3. Auscultatory Signs in Normal Respiration. — 1. **Bronchial Breathing.**—If we auscult the larynx or trachea of a healthy person during inspiration and expiration, we hear a loud aspirating sound which corresponds somewhat exactly with that we can make with the mouth when we put it in position to pronounce *h* or *ch*, and then inspire or expire. We designate this sound as the *laryngeal* and *tracheal*, or by the collective expression *bronchial*, *breathing*, sound. Its peculiarity is its more or less pronounced sharpness (*ch* or *h sound*), and moreover a somewhat rising pitch; again, it is ordinarily somewhat louder (and deeper) in expiration than during inspiration. The sound is formed in the glottis by the eddies which are here formed in the current of air by the sudden narrowing; it is louder in expiration, because the rima glottidis is narrower then than during inspiration. The strength and rapidity of the breathing have a great influence upon the loudness of the sound.

Besides over the throat in front, where the larynx and trachea lie superficially, we hear this sound over the *vertebra prominens* at the back of the neck in healthy persons during moderately strong breathing; also, sometimes, over the upper part of the sternum; very frequently, too, in the interscapular space, and more plainly at the right than to the left of the median line (region of the bifurcation).

Bronchial breathing may be noticed at other parts of the thorax at a varying distance from the above regions during strong breathing, especially with violent, coughing expiration. It is heard earliest over the upper sections of the chest. There may be great individual differences, and yet be within the limits of the normal. Confounding bronchial breathing with the diseased conditions to be mentioned later will be avoided by noting the approximate symmetry of this breathing sound, the condition in feeble breathing, and also the result of the further examination.

A noise which arises in the pharynx and at the lips of the person examined not infrequently disturbs or deceives the beginner: closing the free ear is here recommended.

2. **Vesicular Breathing.**—In healthy persons this is heard wheresoever the lungs lie in contact with the chest-wall (with the exception of in the interscapular space).[1] It is of a very slight shuffling character, resembling the sound we may produce by placing the lips in position to say *f* or *v*. The pitch of this sound can only be approximately recognized (like the clear non-tympanitic sound).

[1] See above.

This sound can only be heard in inspiration, and most plainly at the end of inspiration. In a sound lung *expiration* has a very slight breathing sound which may be said to be of bronchial character. Not infrequently it is wholly imperceptible; sometimes, however, we find inspiration which is simply like a very much weakened vesicular inspiratory sound.

The force of vesicular breathing varies very much. It is most determined by the strength of the breathing: in very strong respiration it is often so loud that it is also heard over the organs adjacent to the lungs, as over the heart, liver, and stomach. In the majority of healthy persons the vesicular murmur is louder upon the left side than upon the right (Stokes). Otherwise, the strength of this breathing sound is determined by the loudness of the pulmonic sound: over thin portions of the lung, as the apices, it is very slight, and likewise it may be weakened by the thickness of the covering, even to such a degree as not to be heard at all. Moreover, there are individual differences which depend chiefly upon the differences in the width of the glottis, also on the elasticity of the chest on the one hand, and on that of the lungs on the other.

Puerile Breathing (Laënnec).—The vesicular murmur in children is remarkably different from that of maturity; the *former* up to about the twelfth year of age exhibits a remarkably distinct, loud, and sharp vesicular breathing sound, which approaches bronchial breathing, especially also in that often it is nearly as strong in expiration as in inspiration. Generally, also, women have a stronger vesicular murmur than men.

Origin.—Since Laënnec different hypotheses have been advanced to explain the origin of the vesicular sound of respiration. As yet there is no unanimity among those who have given attention to the question. Formerly, the explanation of Baas and Penzoldt was accepted. According to this explanation, the vesicular sound represents the laryngo-tracheal breathing[1] sound transmitted through the air-containing lungs. The laryngo-tracheal breathing sound in passing through the inflated lung-tissue is toned down to the fineness and lightness of the vesicular sound. Thus it is quite plausible that vesicular breathing is distinctly audible during inspiration, when the current of air is directed from the larynx and trachea toward the ear, but is almost inaudible during expiration, when the current is reversed. It is, further, very evident that the vesicular sound gives way to the unchanged or slightly unchanged laryngo-tracheal sound, "bronchial breathing," as will be explained farther on, whenever the lung-tissue intermediate between the ear of the auscultator and the bronchi does not contain air in consequence of infiltration (pneumonia), or severe compression (pleuritis exudativa), or where there exists a massive tumor, with compression of the pulmonary tissues between the ear and the bronchial tree. In regard to cavities the conditions are slightly different.[2]

Lately, however, objections have been made to this theory by persons worthy of notice (Dehio, Sahli), which objections necessitate a re-examination of the question. Sahli, particularly, thinks it is beyond all doubt that vesicular breathing is produced entirely by the respira-

[1] See above, p. 121.

[2] See these.

tory movements of the parenchyma of the lungs. We cannot here discuss the pros and cons of the question, but we emphatically cannot, from very weighty reasons, coincide with the authors last mentioned. We consider vesicular breathing to be tubular breathing, which by being transmitted becomes toned down and weakened. But this is true not only of the breathing sound which is conducted from the larynx and trachea, but also of that from the medium-sized, the small, and the smallest bronchi. Every current of air, even in the smallest bronchial branch, causes vibrations and must produce a bronchial sound. This sound, together with that of the larger bronchi, of the trachea, and of the larynx, modified by the air-containing lung-tissue, is what is heard as vesicular breathing; but when slightly or not all altered by solidified lung-tissue or other dense media, it is heard as bronchial breathing of different qualities.

From this it is seen that now, as formerly, we think the explanation of Baas and Penzoldt the more correct one. There remains only to be noticed as an essential modification of this theory that there exists an independent bronchial breathing, even in the smallest bronchi, if air passes through them, and that this bronchial breathing reaches the ear as vesicular breathing.

Sometimes there are special peculiarities of vesicular breathing quite within the normal, which may easily mislead the beginner. During inspiration we may see interrupted or jerking respiration in persons who, at discretion, take deep breaths imperfectly, in a jerking manner; and, further, in whining children, who half suppress their sobs. This kind of jerking breathing exists over all portions of the lungs alike. Moreover, in the portion surrounding the heart, and as far up as to the apex of the left lung, the vesicular murmur exhibits interruptions exactly corresponding to the action of the heart (systolic vesicular breathing, depending upon the unequal entrance of air into this portion of the lung in consequence of the changed volume of the heart, and hence often especially plain in disturbed heart's action).

To learn to distinguish between the bronchial and vesicular breathing is, for the beginner, among the most difficult things in diagnosis. For the comprehension of the latter sound it is strongly recommended always to auscult directly, since the sound is then louder and its nature can thus be more clearly recognized. More than this, it is well to place the ear frequently, for comparison, upon the patient's neck, so as there to hear the bronchial sound.

4. Pathological Sounds in the Respiratory Apparatus.—The following are enumerated:

(*a*) Changes in vesicular breathing.
(*b*) Bronchial breathing, in place of vesicular breathing.
(*c*) The so-called indefinite, transition breathing [broncho-vesicular].
(*d*) Dry râles.
(*e*) Moist râles.
(*f*) Crepitant râles.
(*g*) Friction-sound of the pleura.
(*h*) Succussion-sound of Hippocrates.

From this enumeration, and still more from what follows, it is

evident that the number of pathological sounds to be heard with the diseases of the respiratory apparatus is not small. The chief difficulty is that very often different ones are to be heard at the same time, so that one sound conceals another. It is urgently recommended that the beginner at first practise in such a way that, in auscultating, he endeavor always in the first place to learn to recognize only the breathing sound, and that he then endeavor to direct his attention to other possible so-called accessory sounds (râles, friction-sounds). One can acquire the power to exclude one sound in order to be able more exactly to pay attention to another—to acquire a certain aural dexterity which very much facilitates auscultation.

(*a*) **Alterations of Vesicular Breathing.**—1. The vesicular breathing sound may be *increased in inspiration*, or *sharpened*. This takes place whenever the respiration is increased, as in active deep breathing; in the acme of Cheyne-Stokes' breathing; in certain forms of dyspnea, as of diabetic coma; and where one section of lung is vicariously performing the work of others which have been shut off.

Moreover, it forms a very important sign in bronchitis, here occasioned by the local narrowing of small bronchial tubes in consequence of swelling of the mucous membrane and accumulation of mucus. Not infrequently beginning tuberculosis of the apex is revealed solely by sharpened vesicular breathing in comparison with the sound side, as evidence of accompanying catarrh of small bronchial tubes.

Here the one-sidedness of the sharpened vesicular breathing is of the greatest importance; two-sided sharpened breathing of the upper portion of both lungs almost never has this signification; not infrequently it exists in tightly-laced women, also in children who breathe poorly with the lower portions of the lungs in consequence of a high position of the diaphragm, due to abdominal affections.

2. Vesicular breathing may be *diminished*, either in *bronchial catarrh* in case the entrance of air into a section of lung is notably diminished by the swelling and secretion, or if bronchial branches are more or less closed by foreign bodies or compression. Diminished breathing of a portion of lung is also a consequence of *pleural thickening* and of many conditions which give *pain in respiration*, manifested by the lessened, weakened breathing of the affected side. Diminished interchange of air everywhere, and hence a two-sided extensive weakened breathing exists in *emphysema*, also in *stenosis of the upper air-passages*. All *thickenings of the chest-wall* (tumors, etc., edema) weaken the respiratory sound by rendering the conduction more difficult; and, finally, marked weakening develops rapidly with *pleural exudations*, both on account of the diminished breathing and the more difficult propagation of the breathing sound by the layer of fluid.

In all these cases the breathing sound may even completely disappear; most frequently is this the case with pleural exudations, also in complete *closing of a large bronchial branch*, but it may exist even in *emphysema*.

3. *Prolonged expiration.* This occurs when the exit of the air from the alveoli is more prolonged than is normal, and this condition may be dependent upon diminished elasticity of the lung-tissue: *emphysema* or *bronchitis*—a certain degree of bronchial narrowing, which does not

hinder the entrance of air, but only its exit. Of these two conditions, prolonged expiration is an important diagnostic mark, and here, again, especially comes into consideration bronchitis which accompanies the commencement of *tuberculosis of an apex* of the lung. The prolonged expiration of bronchitis is also generally sharpened, more markedly aspirant, somewhat more distinctly bronchial, than normal. With pronounced bronchial expiration thickening may be conjectured to have taken place.[1]

4. *Jerking inspiration* may likewise be a sign of bronchitis—namely, in case the two conditions are excluded which, within the normal, cause these or a like phenomenon.[2] This pathological jerking respiration, according to its prominence, is confined to the region of the bronchitis, generally to an apex, as in phthisis, and thus is distinguished from the interrupted inspiration of awkward breathing; but it exists always at the beginning of the examination. It results from the delayed entrance of the air into the lung portion of the bronchial tubes if these are narrowed by catarrh.

It takes place with *sharpened* and with *jerking* breathing and *breathing with prolonged expiration*, since in the majority of cases it is called forth or is accompanied by bronchitis—generally, also, toneless râles.[3]

(*b*) **Bronchial Breathing.**—In order to understand the pathological development of this respiratory sound, it first is of the greatest importance that it should be made clear how the respiratory sound normally at the glottis, in the trachea, and in all the bronchi exists as a bronchial sound, how it is further conveyed by the subdivided columns of air in the bronchial tree as a bronchial sound, and how in healthy persons it is deadened by lung-tissue normally containing air into the vesicular breathing sound. *There is no breathing sound without open bronchial tubes; there is no vesicular breathing without lung-tissue containing air.* If between the bronchi and the ear there is no air-containing lung-tissue, if anything at all is heard it is bronchial breathing.

Pathologically, bronchial breathing occurs in *thickening of lung-tissue* of a certain extent—that is, in case it involves an extent that reaches as far as moderately sized bronchial tubes. Here belong acute and chronic *pneumonia*, *infarction*, under some circumstances *new formations*, and also *compression of the lungs*, so that the air is expelled by a correspondingly large pleuritic exudation (this is generally near the upper posterior boundaries), or by tumors of any kind in the chest-cavity, or by very high position of the diaphragm.

In pneumonia, if a considerable number of bronchi become obstructed with fibrinous exudate and mucus, and they thus become completely impassable, they cannot conduct any sound of bronchial breathing. Therefore, in such cases simply no breathing sound at all is heard. It may, however, suddenly appear when, after a fit of coughing, the bronchi have again become passable.

It has been said that complete compression of the lungs by a large pleuritic exudate produces bronchial breathing. Something more must be added to this statement: If the amount of exudation be small, it does not so fully compress the lungs as to expel all the air, but it only produces retraction of the lungs, and the respiration remains

[1] See below. [2] See above, p. 123. [3] See below.

vesicular. However, if the exudation be abundant, it may even compress the bronchi, and that, together with the mass of the liquid, has the effect of cutting off all respiratory sound.[1]

If a pneumonia is combined with a *stopping of the bronchial tubes* (mucus, fibrin), then, on account of this imperviousness, we do not hear anything, but after a cough the bronchial tubes may become pervious: there is bronchial breathing.

Moreover, we hear bronchial breathing over *lung-cavities* and in *open pneumothorax;* and, besides, over the former sometimes, over the latter always we hear it in the form of *amphoric breathing*.[2] It is only when the cavity is near the surface that we have bronchial breathing over it, when it is surrounded by tissue that contains no air and is in open communication with a not too small bronchial branch. In both conditions the bronchial sound really arises from the fact that the air, flowing out of the bronchus that connects with the cavity, or which, connected with a pleural cavity, enters into a larger air-space, or out of this air-space again into a narrow bronchial canal, is set into whirling motion. But there is no doubt that, besides, the sound that is conveyed from the glottis joins with it as bronchial.[3]

In the cases just mentioned the bronchial breathing sound, under various circumstances, may become weakened—namely, either when the advance of the sound to the ear is made difficult or when the breathing is weakened. Thus, in consequence of the fluid which generally lies between the ear and the compressed lung, a slight, distant-sounding bronchial breathing is characteristic of an *exudative pleuritis* ("breathing of compression"); while, on the other hand, in *croupous pneumonia*, almost always there exists a very loud, sharp bronchial sound. But in pneumonia otherwise rare conditions in their turn may weaken the bronchial breathing; in closure of the bronchial tubes, as was mentioned before, we hear low bronchial breathing or else nothing at all; further, in the so-called central pneumonia it may happen that from the part of the lung containing air which lies superficially, a vesicular and, almost concealed by this, a low bronchial breathing sound is produced. Also, the loud pneumonic bronchial breathing may be weakened if the pneumonia is complicated with an *exudative pleuritis*.

In all these cases the low bronchial sound is usually most distinct during expiration,[4] often even only perceptible in expiration as a weak *ch* kind of blowing.

The *bronchial breathing of a hollow space* may be weakened or even lost—weakened in temporary narrowing or closing of the bronchus leading to it by mucus (hence, loosened by cough), or lost by the filling of the cavity with secretion. On the other hand, a thick, callous pleura covering a cavity may rather be the occasion of deadness than that the bronchial breathing is affected.

Special forms of bronchial breathing are the *amphoric* and the *metamorphosing* breathing. The former exists with very large, smooth-

[1] [Compare these two paragraphs with what is said later regarding *increased vocal* fremitus.—*Translator*].

[2] See Amphoric Breathing.

[3] See Amphoric Breathing.

[4] Compare what was said above regarding Expiration.

walled, communicating cavities and in *open pneumothorax*.[1] It is a bronchial sound with metallic tone, exactly analogous to the metallic percussion sound that arises by resonance in large smooth-walled cavities.

Amphoric breathing, moreover, may be found in *open pneumothorax* (and where there is valvular connection), also in *closed*, although more rarely and only very softly, since here the (bronchial) sound of the air flowing into the trachea acquires a resonance in the air-containing pleural cavity; likewise, râles, heart-sounds, may acquire a metallic tone.

Metallic associated sound may also, in rare cases, accompany undefined—that is, bronchial—breathing unnoticeably weakened; thus also, not rarely, in pneumothorax. It might, indeed, be suitable to designate it not as "amphoric," but as "undefined, with metallic associated sound."

Metamorphosing breathing (Seitz). In this the inspiration is divided: it begins distinctly bronchial, like the sound of stenosis, and suddenly changes to a weak bronchial breathing, which is then also heard during expiration. This phenomenon is very rare; it is said to be a sure sign of cavity (?). It is explained that the bronchus leading to the cavity is always first narrowed, and in the second part of inspiration it becomes dilated by the current of air. We too have the metamorphosing breathing only over cavities; but in such circumstances the cavities were always so large that they could be diagnosed also from the remaining phenomena.

(*c*) **Undefined Breathing.**—The breathing sound may in two ways be of such a character that it may be designated either as distinctly vesicular or as distinctly bronchial. First, it may be so *weak* that its character remains indistinct, concealed or drowned by other sounds, particularly by râles; or, while it can be heard, it does not entirely correspond to either type of breathing, but seems rather to stand between the two, thus sometimes inclining more to bronchial, at other times more to vesicular, breathing—"transition breathing," "hinted or indistinct bronchial or vesicular breathing," "sharp breathing with bronchial breath in expiration," etc.

The causes of what is included in the first category are very various.[2] Of course, the examiner's sharpness of hearing is an important factor here. Râles that may be present may frequently be removed or diminished by coughing strongly.

The second group of undefined breathing is, of course, much more numerous with beginners than with those who are practised in auscultation. It is well, however, for the latter also to impose upon themselves some reserve in pronouncing whether it is vesicular or bronchial. The determination is often actually possible either by the tone itself or by the strength of expiration in relation to inspiration. Frequently also, as in beginning phthisis, in various lobular pneumonic deposits, the physical conditions resulting from the pathologico-anatomical changes cause it to appear that there is a "transition breath"—that is, a mingling—in that the infiltrated part of the lung favors the transmission of the bronchial sound unchanged, while the parts containing air convey

[1] See the preceding page.

[2] See what was said above concerning the strength and weakness of the breathing sound.

the breath-sound to the ear toned down to the vesicular sound. Hence, under no circumstances can we miss this idea of "transition breathing," and it is best in such cases simply to describe the breath-sound.

(*d*) **Dry Râles (Rhonchus, Humming, Whistling, Hissing).**—Like all râles, these are pathological sounds. They appear when there is a bronchial catarrh, which furnishes a tough, scanty secretion. They constitute those audible phenomena that are caused by the rushing together of the air and secretion in the bronchial tubes. It is as difficult to make a sharp distinction between a "tough" and a "fluid" secretion of the bronchial tubes as in a stricter sense it is to separate the so-called "dry" from what is later referred to as "moist" râles—much more, since transitions are everywhere present. Meanwhile, however, the class of sounds here referred to take a somewhat special place, both on account of the auditory impression they make and because they exactly correspond to the very toughest bronchial secretions. The humming, hissing, whistling sounds (sonorous, sibilant râles) arise from the fact that the bronchial air-passage is narrowed by the swelling and the mucus, and hence they are sounds of stenosis; but, besides, some of the very fine high hissing and whistling tones may be caused by the presence in the bronchial lumen of tense threads of mucus stretched across, which, like the strings of an Eolian harp, are blown upon by the current of air.

Sibilant râles very often have such a high musical tone that it cannot be deadened even by the air-containing lung. Under some circumstances they may be confounded with the so-called ringing râles [metallic râles].

The *dry humming* often shows unnoticeable transitions to the character of the sound of the moist râles, approaching more nearly to crepitation. According to my view, they may still as dry, become ringing, râles—that is, may exhibit a ringing character like bronchial breathing. This is the case when we have thickening of the lungs and at the same time bronchitis with tough mucus.[1]

The humming, hissing, whistling may be *abundant* or *scanty, loud* or *soft.* It may occupy the whole time of inspiration and expiration and completely conceal the breath-sound, or it may only be heard at the end of inspiration. A very fine soft whistling is sometimes heard during the whole of expiration, since then, so far as vesicular breathing is concerned, the breath-sound is very soft. When they are very loud the sounds may even be heard at a distance (a distinguishing peculiarity of emphysema). Finally, there are buzzing sounds in the chest which may be felt when the hand is applied to it. Cough has sometimes the effect of diminishing, and sometimes of increasing, them—at least the humming is generally very markedly changed by it.

It is not possible easily to confound the humming sounds with pleuritic friction sounds.[2] On the other hand, I have not infrequently found that a very soft, fine humming was mistaken by beginners for sharp, even bronchial, breathing sound. This, as well as the distinguishing of whistling and hissing from a peculiar ringing râle, can only be learned by practice.

Conclusions.—Humming, whistling, hissing sounds, as has been

[1] See ringing râles, under Moist Râles.

[2] See p. 132.

shown above, indicate a dry bronchial catarrh. Spread over the lungs, they are present with *diffuse bronchitis* with *tough secretion*, whether it occurs independently or as an accompaniment of *emphysema*, in which disease they are almost never wholly wanting. In these cases the lower lobes of the lungs are generally the seat of the catarrh. When there is simply bronchitis, then these râles and a sharpened or weakened breathing are the only local physical signs of disease. In emphysema the percussion and auscultatory signs of this condition are also present. Localized dry râles exist as signs of *catarrh of the apices*, which accompanies commencing tuberculosis: here, for example, a low whistling in a perhaps somewhat prolonged expiration may for a long time form the only symptom. Ringing dry râles are rare; they are most frequently heard in pneumonia at the beginning of the second stage.

In all these cases the dry râles may be combined with the moist.[1]

(*c*) **Moist Râles.**—These arise in the bronchial tubes, except the smallest, and in the pathological cavities of the lungs [vomicæ]. Their production requires more or less fluid secretion; the more fluid there is the more moist the sound; if it is tougher, then there are "viscid-moist" râles, a transition to the dry. Generally, the ear directly receives an impression of a greater or less degree of moisture.

Formerly moist râles were explained as being produced by the bursting of bubbles which the current of air caused upon the surface of the fluid. More recently they have received another explanation: according to the analogy of the bubbles which we see formed when we blow through a tube one end of which is immersed in water, it is supposed that the current of air separately moves the air-bubbles which present projections into the bronchial tubes, and that as one such quantity of air breaks the bridge through the fluid and advances, the fluid behind it, immediately rushing on again and occupying the space, shares the vibration in the pent-up air: crepitation râles (Talma, Baas). It is to be added that many consider moist râles in part due to stenosis; and, finally, that it is said that the to-and-fro motion of the secretion produced by the current of air causes râles (Traube). The explanation by Talma and Baas will serve very well for the râles formed in the medium-sized bronchial tubes; for vomicæ it only serves in case the bronchial tubes leading thereto are immersed in the fluid secretion, which, indeed, is ordinarily not the case. Here, and with large bronchial tubes at any rate, we must think of bursting bubbles.

Moist râles may be so numerous that they can be heard in both inspiration and expiration, even outlasting the expiration. If they are scanty, then we are apt to hear them during inspiration, under some circumstances only toward the close of inspiration. A slight cough may increase them, or cause them in case they were for the time being absent.

In cases where the râles are very scanty, scarcely to be heard, it is useful to inquire as to the time of day the cough is the most frequent, and to listen to them before the occurrence of the paroxysm of cough, so as to make the examination before the bronchial tubes have been cleared of mucus (as shortly after waking).

[1] Regarding these see below.

The different moist râles make the impression upon the ear of being of different "sizes," and even beginners can without difficulty judge approximately whether they are found in a large or a small bronchus or cavity; we speak of *large*, *small*, also *medium-sized* râles. The discrimination of râles in this respect is very important; for instance, we may distinguish whether we have a bronchitis of only the large, or whether the smaller, tubes have become involved; dangerous capillary bronchitis is manifested by very small, fine râles, and also by crepitant râles.[1] Large râles may furnish an index in the examination of the apices: these contain only very small bronchi; hence, if in an apex there are large or only medium-sized râles, these cannot arise from the bronchi there; hence there must be a pathological space—a cavity. If there are *large râle-sounds* which undoubtedly arise in the *apex*, they are a most certain sign of cavity.

The *loudness* of the râles does not depend upon their number, but upon the strength of the breathing. But the loudness furnishes an indication of the place where they arise: *cæteris paribus*, the sound will be loudest at the point where the ear is nearest to them. It may be of the greatest importance to locate them exactly. Here, again, the most striking example concerns the diagnosis of phthisis, and, too, of the ominous catarrh of the apex. By a superficial examination it may easily happen to the inexperienced, especially in the examination of the back, that he locates râles which come from the neighborhood of the root of the lungs, and are those of a benign bronchitis, in the apex, and hence makes the diagnosis of phthisis.

It is of the very highest value, but often not easy, to distinguish whether we have a ringing or ("consonant," Skoda) or a non-ringing râle-sound. The former is acoustically related to the latter, as the bronchial breathing sound is to the vesicular (as tympanitic percussion note to lung-sound), and, like that, ringing râles appear if there be present either a *thickening of the lung* of sufficient extent or if there be a *cavity*. But yet bronchial breathing and ringing râles, and vesicular breathing and non-ringing râles, are not always necessarily associated together; thus, not infrequently when there are small cavities, and even large ones, especially in the lower lobes, in case they are covered by a not very thick layer of air-containing tissue, we hear ringing râles when the breathing is undefined, yet hinting toward the vesicular. In children, even when there is no trace of cavity or thickening, in simple bronchitis the râles may reach the ear as loudly ringing (from the pronounced elasticity of the lungs and of the thorax). On the other hand, in pneumonia and pleurisy we sometimes hear bronchial breathing and non-ringing râles.

But now, corresponding to "transition breathing," very frequently there are to be heard such râles as stand between the non-ringing and the pronounced ringing ("hinted" or slightly ringing râles). It is often difficult to interpret these. In general, with children they furnish no reason for the supposition of thickening or cavity more than with adults.

Loud ringing, hinted ringing, and non-ringing râles are often found together; we may even say that almost never do we hear ringing râles

[1] See p. 131.

alone at one place. But, of course, if they are present they predominate. Though they exist very near together, yet they can be locally separated, as sometimes in *emphysema;* here, with extensive humming, whistling, and non-ringing râles at a certain point of the lower lobe, there may be ringing râles (perhaps without bronchial breathing and without deadened or tympanitic resonance): this makes a bronchiectatic cavity probable. But, also, by the same signs, in general bronchitis a broncho-pneumonic deposit may be made known.

As the ringing râles correspond to bronchial breathing, so in their manifestation the so-called metallic râles correspond to *amphoric breathing* (metallic percussion-note); but again in such a way that the two symptoms are not necessarily associated together. The metallic râles then occur in correspondence with very large, smooth-walled, superficially-located cavities, and also in *pneumothorax*, where, arising from sections of the lungs which are breathing (even if on the other side), they are to be regarded as râle-sounds in the air-containing pleural cavities endowed with resonance.

Sounds of falling drops. These are often only separate, generally very much inflated, moist râles, which have a high metallic note; sometimes, indeed, there is only one in each phase of the breathing; then the above-mentioned designation of it serves.

Water-whistling, or the sound of lung-fistula (Unverricht, Riegel). We thus designate a metallic râle or very fine metallic gurgling or splashing which occurs in open pneumothorax if the patient's position is such that the opening in the pleura is directly below the smooth surface of the fluid, and if the patient then draws a breath (first observed by Unverricht while puncturing and aspirating a case of *hydropneumothorax*).

(*f*) **Crepitant Râles** (**Crepitation**).—Briefly expressed, by this we understand the finest râle sounds. It occupies a special place on account of its acoustic peculiarity, on account of its cause, which permits its classification either under the moist or under the dry râles, and, finally, on account of its special diagnostic meaning.

The so-called *atelectatic crepitation* occurs in health, and still more in disease, over parts of the lungs which have for a time been breathing poorly and now are again distended by a full breath. Most frequently is it observed after quite long, especially low, dorsal position, over the lower parts of the lower lobes. It is purely inspiratory, and generally disappears after the first deep respirations.

Like this are crepitant râles which are to be heard in *croupous pneumonia* during the first, and in the beginning of the third, stage (*crepitatio indux and redux*), sometimes in *catarrhal pneumonia*, moreover in *pulmonary infarction*, generally speaking in all kinds of consolidation, and, finally, especially in *edema of the lungs*.

In all these cases we have to do with *crepitation heard during inspiration*, or, at most, only the beginning also of expiration, which occurs in very fine and equal-sized bubbles. It is well compared to the noise produced by rubbing a lock of hair between the fingers in front of the ear, or by separating the thumb and finger moistened and pressed together as they are held before the ear (Eichhorst). It arises in the smallest bronchial tubes, the alveolar spaces, and in the alveoli

when these are collapsed and glued together or partly filled with secretion, and then during strong inspiration their walls are torn apart or freed from secretion. It is only in individual cases that this crepitation is heard in expiration and *only* in expiration (Penzoldt).

The *non-uniform crepitation*, so called, forms the transition from these sounds to the fine bubbling râles. More than elsewhere it occurs with *capillary bronchitis* and also in *edema of the lungs.* It is to be understood as a mixture of peculiar crepitations and small bubbling râles, and accordingly, in its coarse sounds, it is to be heard also in expiration.

(*g*) **Pleuritic Friction-sounds.**—The respiratory gliding of the pleura costalis upon the pleura pulmonalis, which normally is noiseless, is perceived by the ear, and can also be felt when the hand is laid upon the chest, when there are inflammatory deposits upon the serous surfaces. Thus it is really the *pleuritis sicca* that causes it. Only in rare cases of unevenness of the pleura is this phenomenon observed in the absence of inflammation, as in acute *miliary tuberculosis* of the lungs and pleura; also in *pneumonokoniosis.* The conditions most favorable for the occurrence of this sound are where the respiratory movement of the lungs (forward and downward) is most marked—below and especially at the sides. But this sound may also exist farther up, even almost as high as the apices.

Pleuritic friction-sounds are like regular scraping or like a scratching, creaking, beginning in distinct pauses, which ordinarily are louder during inspiration than expiration. Not only can they be heard, but they can be felt, only weaker: "palpable friction-sounds," best recognized by the laying on of the flat hand. This friction-sound is not changed by cough, but continued deep breathing often causes it to disappear, since in this way the unevenness, upon which it depends, is smoothed out.

When this friction-sound is very loud and characteristic it is easily recognized. A difficulty may arise when it is very softly heard; this often occurs from the fact that the examiner does not auscultate at the right spot, for friction-sound is heard in only a circumscribed area, since it is poorly transmitted. A further difficulty lies in distinguishing it from certain medium-sized, tough, moist râles (cracking râles) and from soft buzzing; here it is most important to take note of the character of the particular sound, and the knowledge and recognition of this can only be acquired by practice. We may make use of the effect of coughing as an aid. Sometimes moderate pressure with the stethoscope increases the pleuritic sounds; also, palpation may help us to recognize them. Râle-sounds are seldom, or, at most, only slightly to be felt. It is to be remembered that friction- and râle-sounds may occur at the same time. Besides, in pneumonia, I have observed this most frequently in disseminated tuberculosis and in caseous pneumonia of the lower lobes.

Friction occurs with all kinds of pleuritis. It occurs (seldom) in acute exudative pleuritis in the beginning of the attack, and also later as a favorable sign with the absorption of the fluid exudation. There can be no friction-sound while there is fluid present, since it is only heard when the pleural surfaces are in contact. In *chronic pleuritis* it

may last indefinitely and over a large extent. Of the diseases of the lungs which usually are accompanied by *pleuritis sicca*, many are first revealed by the friction-sounds which the latter causes: thus, *phthisis*, also *pyemic deposits in the lungs*, *infarction*, *bronchiectasis* with reactive *pneumonia*, and *pleuritis with emphysema*.

Regarding pleuro-pericardial friction-sounds (pericarditis externa), see under Auscultation of the Heart.

(*h*) **Hippocratic Succussion.**—This is a phenomenon very easy to understand:

In *sero-* and *pyopneumothorax*, after a strong shaking of the chest, as in any vessel partly filled with fluid, there is splashing. This splashing, through the resonance associated with metallic tone, like all the audible phenomena of pneumothorax, is heard at a distance or by applying the ear to the chest.

This sign is usually most distinctly manifest when there is a small effusion and when it is serous. It is almost pathognomonic of hydro-pneumothorax in that it only elsewhere occurs in very isolated cases of large cavity with quite fluid contents.

The direction of Hippocrates was to shake the patient by the shoulders, but, on account of the grave condition of most of these patients, the greatest care is necessary. Many quickly learn to shake the body so as to produce the sound themselves.

Confounding this with the splashing from the stomach or colon will be avoided by local examination of these organs and by repeated examinations.

Palpation of Vocal Fremitus (Auscultation of the Voice).

Strictly speaking, this method of examination belongs in part to Palpation and in part to Auscultation, but at the same time it has a place here, because this comes next in the course of a thorough examination of patients. It is, besides, of sufficient importance in itself to be treated separately, because, after Inspection, Palpation, Percussion, and Auscultation have been completed, not infrequently it happens that this casts the decisive vote.

The vibrations of the glottis in phonation (speaking, singing, screaming) originate in the column of air in the trachea and bronchial tubes rather than in their walls; they traverse the lung-tissue, where, in case this is normal, they become considerably weakened, then the wall of the thorax and its coverings, and may be felt by the hand laid upon the chest as a whizzing: voice vibration, voice fremitus, pectoral fremitus (besides heard as indistinct *humming*).[1]

The technique of this method of examining is as follows: While the patient counts aloud, say from thirty to forty or from ninety to one hundred, the hand is laid upon different parts of the chest. Generally we employ the palm of the hand, but for finer examination it is preferable to apply the ball of the little finger or the tips of the first, second, and third fingers. Practice of the method last mentioned enables one to dispense with auscultation of the voice. Differences of voice-vibration are distinguished by comparison of different locations, and particularly

[1] See p. 135.

of symmetrical points. It is quite unnecessary in making this comparison to apply both hands at the same time to the two sides of the chest; the difference is much more distinctly felt if we examine with the same hand, first upon one side and then upon the other.

In the case of female patients with a very soft voice or of children it is better not to have them count, but to continuously pronounce the palatal *r*. (Compare the following paragraph.)

Within normal limits, vocal fremitus is stronger the stronger the voice; it is very distinct when the voice is rough and deep, weak if the voice is high, and even not to be felt at all when the voice is high and thin (light), as is sometimes the case in women and children. The separate vibrations are felt more distinctly the richer and more prolonged they are. The fremitus is stronger upon the right side of the chest than the left, probably because the right bronchus is the larger in diameter. It is, moreover, very noticeably influenced by the thickness of the covering (muscles, mamma, subcutaneous fat).

There may be pathological conditions present upon one side that will not propagate the vibration of the voice so well as a normal condition would do, which may diminish or remove the vocal fremitus; on the other hand, they may better propagate it—strengthen the vocal fremitus.

Weakness or *suppression of vocal fremitus* occurs with *pleuritic exudation* (on account of the narrowing of the bronchial tubes from compression and on account of the encroachment of the fluid); with *pneumothorax*, on the one hand, either on account of the poor conduction through the bronchial tubes of the retracted or the compressed lung, or, on the other, because it is not conducted through the air-cavity. (If, however, there should be growths on the pleural surfaces, even if only in the form of fine fibers, these ordinarily act as good conductors of vocal fremitus.) Finally, vocal fremitus is weak or suppressed with tumors of the pleura and all *thickenings of the chest-wall* (abscess, edema), and in *closure of the bronchial tubes*, since these are the most important means of propagating the oscillations (closure from mucus, masses of fibrin, foreign bodies, compression).

Increase of vocal fremitus is observed—in pneumonia, since the solidified lung-tissue is a better conductor than when it contains air; for the same reason, sometimes when the lung is compressed against the thorax-wall; above pleuritic exudation and generally posteriorly at the roots of the lungs; and in cavities with open bronchus and small secretion, here partly by the good conduction of the sound and partly by consonance.

Vocal fremitus is an extremely valuable means of distinguishing between pneumonia and pleuritic exudation. Yet it may, in rare cases, so far deceive as that in pneumonia, if the bronchial tubes are stopped by secretion, there is no increase of vocal fremitus; it is even diminished, and, occasionally, with complete filling-up of the bronchial tubes, may even disappear altogether. Under some circumstances after cough and expectoration, as after a cool bath, it may return. It is easy to see how various the result may be if pneumonia and pleurisy, or if a cavity and thickened pleural walls, are combined.[1]

[1] Compare this paragraph with p. 126.

In most cases, in my opinion, *auscultation of the voice* may be dispensed with where one is thoroughly trained in testing the vibration of the voice by palpation especially by using the tips of the fingers. It is of value sometimes in the case of stupefied patients and children who cannot be induced to count. In reality, its result is fully analogous to that of palpation. Normally, over the thorax we hear the voice of the person examined as an indistinct humming, which pathologically may be weakened or lost; but it may be strengthened to an extraordinary loudness (*bronchophony*), wholly under the conditions which correspond to those that influence vocal fremitus.

We sometimes find a very marked bronchophony over those cavities where we hear amphoric breathing and metallic râles. Here, also, the bronchophony may acquire a kind of metallic quality (*Laënnec's pectoriloquy*.

Egophony, "bleating voice," is a peculiar nasal, bleating pectoriloquy sometimes heard, with pleuritic exudations, in the neighborhood of the upper boundary of deadness.

Auscultation of the whispered voice was introduced by Baccelli. He found that it was propagated by serous exudations of the pleura, but not by purulent, since the latter dispersed the sound-waves. In most cases this method must be considered as without value, since in large serous exudations with marked compression of the lungs we as often do not hear the whispered voice. We may recognize it in very small and fresh purulent exudations unconnected with thickening of the pleura.

Palpation and auscultation of the voice of course cannot be made in all those cases where the voice cannot be produced, as in unconsciousness and exhaustion, or when the patient is dumb (aphonic), or where, from caution, we do not wish to have the patient speak aloud, as in hemoptysis, peritonitis, etc. Scherwald has recently devised a new procedure, which can be recommended—*plegaphonia*, or auscultation during percussion upon the larynx or trachea. The vibrations produced in this way take the place of those of the vocal cords during phonation, and this procedure is exactly synonymous with auscultation of the voice.

Mode of Application.—We have some one else place a large ivory or hard-rubber pleximeter upon the surface of the thyroid cartilage or upon the trachea, and percuss with a hammer (sometimes the patient himself can do both). The patient closes his mouth. By preference we auscult during expiration. Ausculting on the thorax, we hear the blows—1, over the sound lung, very markedly weakened (loudest over the apices), as if it were vanishing, not tympanitic, but with a cracked-pot sound; 2, over infiltrated lung, very loud, tympanitic, with Wintrich's change of sound; here, also, the ear has a sensation as if the blows were upon itself; 3, over an exudation, simply weak even to complete absence; 4, over cavities, the same as over infiltrated lungs; over large open cavities, very loud, "smiting;" 5, over pneumothorax, a metallic sound.

On the reverse principle Gabritschewsky[1] has constructed a pneu-

[1] *Berl. klin. Wochenschrift*, 1890.

matoscope. He auscultates the percussion of the thorax from the mouth. We have had no experience with the method.

EXPLORATORY PUNCTURE OF THE PLEURA; DIAGNOSTIC STUDY OF THE FLUIDS OBTAINED BY PUNCTURING.

1. Exploratory Puncture.

Mode of Procedure.—For this small operation we employ either an ordinary large hypodermic syringe, or, better, a somewhat larger syringe holding two grammes, of the same construction, with a steel cannula somewhat stronger and about seven centimeters long. The instrument must of course be carefully disinfected before it is used, both for the safety of the patient and from the diagnostic standpoint. For making cultures and for vaccination experiments the whole syringe, but particularly the piston, must be absolutely free from germs. This can be approximately obtained in a syringe with an asbestos piston, which is very much used lately. Absolute security is not obtainable with these, but all other instruments which have been mentioned, and which can be perfectly sterilized, are too unpractical or too expensive. With the piston pushed in the needle is inserted in an intercostal space perpendicular to the surface, and then the piston is withdrawn. If the point of the needle rests in fluid, this will rush into the syringe.

Directly before making the exploratory puncture the patient must be placed in exactly the same position he is to occupy during the operation, then be carefully examined and especially percussed.

In this way we may ascertain whether there is fluid in that portion of the thorax, and of what kind it is. It is especially applicable in the diagnosis of pleuritis (more rarely in hydrothorax and hydropneumothorax). It is to be performed in the following cases:

1. *When there is the slightest doubt whether there is pleuritis or not.* In the first place, we have to consider the differential diagnosis between *pneumonia*, *tumors* of the chest-cavity, and *thickening of the pleura*.[1] In these latter conditions the syringe will draw out nothing at all, or, at most, only a drop of blood. If the exploratory puncture yields a positive result, it has of course a definite diagnostic significance. If, on the contrary, it is negative, that does not positively prove that there is no liquid in the interior of the thorax. In the first place, one may have missed the fluid with the point of the needle. This easily happens when there is only a slight amount of extravasation imbedded between thickened tissue. Again, sometimes no fluid is obtained because it is very thick or contains flocculi of fibrin, and this of course is more apt to be the case the smaller the cannula used.

With reference to these possibilities it is often advisable to repeat the exploratory puncture one or more times. It is important not to be discouraged by a fruitless puncture if there is suspicion of a secluded empyema. The operation, which is quite harmless if performed correctly, can be repeated four or more times at one sitting.

2. *To determine the nature of the fluid contained in the pleural cavity.* If the fluid withdrawn is quite or almost clear like water, if it contains

[1] Compare pp. 112 and 134.

no material elements, if there is no effusion of fibrin and it contains only very little or no albumin, then the fluid is a *transudation;* otherwise it must be regarded as an *exudation.* Again, the exudation may be serous, sero-fibrinous, sero-purulent, chylous, or chyliform, and, finally, it may be hemorrhagic. The purulent extravasations are either odorless or stinking, and in the latter case are ichorous or feculent.

The chylous and chyliform extravasations are similar to the purulent; they are, however, different from the latter in having a more milk-white color, and often also by accumulation of fat on the surface. The chylous extravasations take their origin from communications of lymphatic vessels or of the ductus thoracicus with the pleural cavity, as when they burst from over-distention (filaria disease). The chyliform extravasations are transudations or exudations consequent upon carcinosis or sarcomatosis of the thoracic cavity. These extravasations contain great quantities of tumor-cells from fatty degenerations. Chylous and chyliform extravasations microscopically are very much alike; chemically, they differ only by the circumstance that the chylous contains a demonstrable amount of sugar, while in the chyliform there is no sugar unless complicated with diabetes.

The *microscopic examination* always reveals some pus-cells in the serous and sero-fibrinous exudations. There are all stages of transition, from the serous exudate containing a minimal amount of cells to that which shows macroscopical, as well as microscopical, purulent contents; but in practice it is only seldom that one is in doubt whether a fluid should be designated as purulent or sero-purulent. Transition forms are to be called sero-purulent. Moreover, it is to be noted that many old sero-purulent and purulent exudates have the peculiarity that they deposit a sediment within the chest-cavity, so that in the dependent parts of the pleura there is thick pus; in the higher parts there is a more or less pus-containing fluid. In making an exploratory puncture in cases where the exudate has existed for a long time, especially in patients who have maintained a certain position for a long time, it is important to remember these facts and to puncture low down as well as high up on the thorax.

The chylous and chyliform extravasations contain great quantities of fat-corpuscles, fat-cells, fat-containing leukocytes, and endothelia. In the chyliform fluid there are sometimes found carcinomatous and sarcomatous cells. However, it is only very seldom that a diagnosis of carcinomatous and sarcomatous pleurisy can be made from the quality of the exudation. There occur here serous, hemorrhagic, and the chyliform extravasations which have just been mentioned. A positive diagnosis is never possible except when particles of the tumor—as, for example, when groups of cells in characteristic arrangement—are mixed with the fluid. Single cells do not furnish a positive indication. Considerable lumps consisting of cells of very different form and size, which give with iodin the glycogen reaction, are more particularly significant of carcinoma.

In old pyemic pus and in extravasations which contain much fat cholesterin is often found in the form of the well-known plates (see Fig. 48).

The *microscopical examination for micro-organisms* by means of

the cover-glass preparations of the fluid and its sediment under all circumstances is an imperfect method. However, it gives the physician some conclusion, especially in purulent exudates, and sometimes even in serous, and hence it ought not to be omitted, especially where prompt action is required. Several cover-glass preparations should be made at the same time—one with simple anilin coloring, one with Gram's, one with the stain for bacillus tuberculosis. It not infrequently happens that these preparations furnish a picture which is confirmed by cultures. For instance, by the method of Gram only diplococci, or only staphylococci, or the tubercle bacilli (the latter very rare) are met with, or there are great quantities of different bacteria (ichorous exudate).

The examination for actinomyces is only made by the microscopical examination of the granules: to make cultures is difficult and unnecessary.

But if one wishes to make a careful diagnosis with reference to the presence of micro-organisms, cultures and vaccination experiments are necessary. The latter are particularly advisable when tuberculosis is suspected, because by vaccination we may get a positive result, though the culture process may have been negative. Vaccination is made upon guinea-pigs.

The *results of the examination for bacteria* are in general the following:

Transudations are always found free from micro-organisms.

Serous and sero-fibrinous exudates are likewise sometimes free from them, but more frequently the following micro-organisms are found in them: 1. The staphylococcus pyogenes albus: pleuritis after typhoid fever, after croupous or broncho-pneumonia. 2. The streptococcus pyogenes: various kinds of pleurisy, and also in abdominal affections. 3. Fränkel's pneumococcus: metapneumonic exudations—that is, such as follow croupous pneumonia. 1 and 3 may also occur together. It is interesting to note, as was first shown by Levy, whose observations have been corroborated several times by us, that serous exudations need not necessarily become purulent from this species of cocci, but, on the contrary, may disappear without becoming purulent. 4. Bacilli of tuberculosis are almost never found in the pleurisies of tuberculous patients.

Empyemas may be entirely free from micro-organisms—a circumstance which very strongly points to tuberculosis. Moreover, they may contain—and this is generally the case—the staphylococcus pyogenes albus, aureus, citreus, or the streptococcus pyogenes, or several of these cocci together. Empyemas containing only staphylococci not infrequently take a benign course. Metapneumonic emphysemas mostly contain Fränkel's diplococcus, and very often only this, but sometimes also Friedländer's pneumococcus; and under some circumstances there may be, besides Fränkel's diplococcus, staphylococci and streptococci. The metapneumonic emphysemas with Fränkel's diplococcus usually contain very thick mucous pus, which it is difficult to aspirate. They have a remarkable tendency to be spontaneously absorbed.

Ichorous and feculent-ichorous exudations are always distinguished

by great quantities of micro-organisms, among which streptococci and staphylococci are never missing.

A rare but very significant constituent of purulent pleuritic exudations, also of peripleural abscesses, is the *actinomyces*.[1] However, the granules which this fungus forms do not pass through the cannula of the ordinary hypodermic syringe unless they are very small. Granules of considerable size require the use of the larger syringe already mentioned or a thick trocar. Therefore, when there is a suspicion of actinomyces reliance cannot be placed upon a fine syringe, which may aspirate pus without organisms or not aspirate anything at all.

Hemorrhagic exudation makes the existence of *tubercle* or *carcinoma* of the pleura probable. If the exudation is *fœculent*, there is some connection with the intestine. But sometimes there is no disease of the pleura at all, but a *diaphragmatic peritonitis*[2] which simulates a pleuritis.

Exploratory puncture, finally, must always be made—

3. *Before operative procedure* when pleurisy has been diagnosed, even if the diagnosis seems to be perfectly certain.

From what has been said it is clear that we operate by preference, but by no means exclusively, upon the lower parts of the thorax. Of course we must avoid the region of the heart, and when there is a suspicion of aneurysm explorative puncture must be omitted; otherwise there is no need of anxiety. When the exploratory puncture is made with the observance of every possible precaution it is not a dangerous procedure. The puncture is made quickly, in an intercostal space, as far as the needle will reach; if nothing is obtained, the needle is slightly withdrawn and suction again made. We may sometimes puncture at several points in succession.

2. Chemical Examination of Aspirated Fluid.

The greater quantities of fluid which are obtained when aspiration is done from therapeutic reasons may be used for chemical examination. This is chiefly the determination of the percentage of albumin, which has hitherto been made for diagnostic purposes in cases where the question was one of differential diagnosis between serous exudations and transudations. In the former the average amount of albumin is 4 to 6 per cent., in the latter about 2 per cent. But, on the one hand, in severe hydremia inflammatory exudations may be poorer in albumin, while, on the other hand, transudations may contain even as much as 3 per cent. of albumin (Citron). Therefore the percentage of albumin has very little diagnostic value. Even later examinations and experiments have not changed this observation.

In order to simplify the determination of albumin, Reuss has given a formula for the direct approximate determination of albumin from the specific gravity. The formula is as follows:

$$A = \tfrac{3}{8}\,(\text{S. G.} - 1000) - 2.80.$$

A = albumin in percentage; S. G. = specific gravity at 15° Celsius.

This formula has the fault that the specific gravity does not depend

[1] See below.

[2] See this.

upon the percentage of albumin alone, but also on the other dissolved constituents of the fluid. Moritz estimates the average error which is caused by that to be only ± 0.157. Contrary to him, Citron has found the formula to be unreliable in a much higher degree. We ourselves had the same experience years ago, and therefore cannot recommend the calculation by Reuss's formula. Runeberg has somewhat changed the formula, but in no essential way.

For differentiating between chylous and chyliform extravasations chemical examination cannot be omitted. The presence of distinctly demonstrable quantities of sugar indicates a chylous extravasation —*i. e.* a communication between the pleural cavity and the ductus thoracicus.

METHODS OF MEASURING AND STETHOGRAPHY.

Measuring the Thorax.—A single measurement serves to determine the size of the chest, and to secure an approximate point of departure for determining its relation to the development of the rest of the body; but it does not furnish knowledge of diseases any better than, with sufficient practice, is given by inspection and palpation.

On the other hand, it has a very great value in connection with tracing the cross-section of the chest upon paper if it is employed to determine the changes which the chest undergoes in the course of a certain disease.

We measure the diameter of the thorax with the caliper-compasses, and it is best to take the broad diameter at the highest point of the axilla, the deep or sterno-vertebral diameter on the level with the nipple and the insertion of the second rib. In tracing a cross-section of the thorax upon paper we must, of course, make the transverse and antero-posterior diameters at the same level (whether at the nipples or lower down). The circumference of the breast is generally measured at the level of the nipple, but sometimes over the highest points of the axillæ and at the lower end of the corpus sterni. The length of the chest may be ascertained by measuring in the mammillary line from the clavicle to the border of the ribs. The linea costo-articularis is very useful for determining any change in the length.[1]

The delineation of the form of a cross-section of the chest is made in the following manner: The opposite diameters at a given point are measured and are marked upon a sheet of paper. Next a lead hoop or wire is accurately fitted first to one and then the other side of the chest at that level, carefully removed and traced upon the paper. Instead of the leaden hoop (which is entirely satisfactory), we may employ Woillez's cyrtometer, which is a chain with links that move stiffly.

Frequent measurements of the diameters and circumferences, as well as tracing the cross-section in the course of disease, may give not unimportant results: in determining an increase or diminution of the quantity of pleural exudate or of the progress toward recovery by the amount of shrinking; in retraction of the lungs; but especially in

[1] See under Spleen.

all kinds of tumors of the chest-cavity. Thus, where aneurysm is suspected or a mediastinal tumor, the slightest increase in the antero-posterior diameter or of the circumference of the chest is of great significance.

In view of what has been said, the statement of the exact measure is impossible. It is only important to know that the right side of the chest, in people who are right-handed, measures about 1 to 1½ cm. more than the left; also, that the circumference of the chest at the level of the nipples in healthy persons is increased in inspiration about 5 to 7 cm.

Spirometry, Pneumatometry, and Stethography.—The value of these methods of examination for diagnosis is very limited. The latter two could even be omitted if they did not sometimes find application in judging of the course of certain diseases.

Spirometry is employed to ascertain the vital capacity of the lungs —that is, the quantity of air which, after deepest inspiration, can be given off by the deepest expiration. This is done by means of a Hutchinson's spirometer, which is constructed on the principle of a gasometer. The quantity of air varies in such a degree not only with age, sex, weight, and physical vigor, but also with undefinable individual circumstances, and to such an extent that ascertaining it but once has very slight diagnostic value. The relations of the size of the body to the vital capacity of the lungs are relatively the most constant. Von Ziemssen found that in men, if to each cm. of stature there was less than 20 c.cm. of vital capacity (or, in the case of women, less than 17 c.cm.), there was either probably a considerable disturbance in the organs of respiration (phthisis, emphysema, adhesive pleuritis, bronchitis) or it already definitely existed. On the other hand, where the relation was as 1 : 25 (or 1 : 22) this was improbable. The vital capacity is of more importance for supplementing other methods of examination in the course of observation of a patient, for the reason that it changes with the recovery from, or exacerbation of, the given disease. It is to be observed that there seems to be an increase in the vital capacity of every patient in consequence of increased practice. Spirometry does not here have an independent value.

Pneumatometry is the determination of the pressure of the respiratory air during inspiration and expiration. It is determined by means of the pneumatometer of Waldenburg, improved by Biedert and Eichhorst, a modified mercurial manometer. We find that in health the expiratory pressure is always greater than the inspiratory, but the absolute results vary still more than those obtained by spirometry. The diminution of the expiratory pressure in emphysema is important, and furnishes a certain conclusion as to the severity of the disease, as well as of improvement or extension. Diminished inspiratory pressure in stenosis of the air-passages, in phthisis, and in exudative pleuritis has no diagnostic meaning.

Stethography is the graphic delineation of the respiratory motions of the chest and of the diaphragm. Different forms of apparatus have been constructed to represent graphically thoracic and diaphragmatic respiration—among others Riegel's double stethograph, Marey's pneumograph, Knoll's rubber bottle for epigastric respiration. But as such

apparatuses are not necessary for clinical diagnosis, we omit any description of them.

COUGH AND EXPECTORATION.

Cough is caused in the following way: By the closure of the glottis after a deep inspiration has been taken, the pressure in the thorax by means of the auxiliary muscles of expiration is increased, and then suddenly the glottis is opened; there results an audible outrush of air, which in turn brings with it the substances forming the expectoration (which substances cause *râles*).

The ability to cough is lost not only when the crico-arytenoidei laterales muscles in the larynx, but also the respiratory muscles, are paralyzed (bulbar paralysis). Pain, also, may cause suppression of cough.

Cough may be *spontaneous* or *reflex*. Reflexive cough-irritation may arise from all parts of the mucous membrane of the larynx, trachea, and bronchial tubes, as well as from inflamed pleura ("pleural cough" no doubt occurring not infrequently). The trachea is especially irritable, and particularly the region of the inter-arytenoidean space, likewise the bifurcation; inflamed mucous membrane is more irritable than normal. There is never any irritative cough from the lung-tissue.

Cough may also arise reflexively from the stomach and sexual organs of women. Here belongs the dry, short, and slight cough of many women at the approach of menstruation, as well as the cough of hysterical subjects.

The cough which is caused by disease of the respiratory organs at the points above mentioned is either caused by accumulation of secretion, or more seldom by foreign bodies in the air-passages, or by inflammatory processes, by tumors, irritation from pressure, which may be in the air-passages or the pleura. In the latter case the cough is of course dry; but if due to increased secretion, the peculiar accessory sound varies with its character, whether it be more moist, more fluid, or more coherent.

Cough is thus a most important sign of disease. Moreover, in spite of the existence of irritation, a patient whose mind is markedly obtunded (as, for example, in typhus abdominalis [typhoid fever], in disease of the brain, in carbonic-acid poisoning, in the death-agony, etc.) may not have any cough; hence, in these cases there is often considerable mucous rattling in the trachea, without any expectoration. The sudden stopping of cough and expectoration in consequence of unconsciousness, often accompanied with weakness, is therefore, in many diseases of the lungs, as in pneumonia, a bad sign; in phthisis, also, it sometimes denotes approaching death. It has already been mentioned that cough may disappear as a result of paralysis of the muscles concerned in coughing.

We can draw no positive diagnostic conclusion from the frequency of the cough. Regarding the time of day when it is most apt to occur, frequently in phthisis, and also in chronic bronchitis, this regularly occurs soon after waking.

Dry cough is generally weak. The ominous dry cough of consumptives, which probably not infrequently originates in irritation of the pleura, is well known, and also the dry pleural cough in commencing pneumonia, which is suppressed on account of the pain in the chest. In a most striking manner this latter cough can also often be observed in aspirating the thoracic cavity. But dry cough, as has been mentioned above, is occasionally also a reflexion from abdominal organs, and, finally, it is sometimes simply a bad habit.

There is a *cough with tough expectoration*, difficult to be dislodged, generally brought up after a long series of labored efforts; at the end there usually is hawking; the patient often pauses to rest, and then continues to cough until a final hawking and expectoration, as in emphysema with tough bronchitis and in croupous pneumonia.

Moist cough with fluid (more purulent) expectoration is easier, "looser." Here it is often remarkable what a quantity of sputum is thrown off, as from a cavity—sometimes from two efforts at coughing. Moreover, with patients who are weak and very miserable often a series of efforts are necessary, which efforts then generally end with hawking (phthisis *in extremis*).

In *whooping-cough* the cough occurs in pronounced paroxysms. Here the inspiration is noisy, because it must be taken as quickly as possible, and also because the glottis is narrowed by swollen mucous membrane. In consequence of the prolonged effort at coughing, of the constantly increasing intrathoracic pressure, and the diminished breathing, which causes a disturbance of the interchange of gases and blood-stasis, there is cyanosis; here, as otherwise in long-continued labored efforts at coughing, especially in phthisis, very frequently they finally end in vomiting. Severe attacks of coughing, moreover, result from swallowing the wrong way, as in paralysis of the throat from various causes. Unconscious patients often swallow the wrong way without any cough.

The tone of the cough, like the voice, may be unnaturally deep and rough in ulceration of the larynx; in stenosis of the larynx it is either a short stenosis sound or rough and bellowing (the latter with children with diphtheria or false croup); in continued aphonia the cough is sometimes toneless, sometimes remarkably rough and sharp.

Hawking only brings up masses lodged in the pharynx, larynx, or the upper part of the trachea; but it must not be understood that what is thus brought up is formed at these locations: it may be brought to the lower part of the larynx by previous cough or by the motion of the ciliated epithelium of the trachea.

Expectoration, Sputum.

By the term *expectoration* is understood all those substances collectively that are brought up from the air-passages by coughing and hawking. According to the existing disease, they are formed from the secretions of the laryngeal, tracheal, and bronchial mucous membrane, from the contents of the alveoli of the lungs, and, lastly, from the contents of pathological cavities of the lungs or from the lung-tissue. In rare cases purulent exudations from the pleural cavities, from rupture

of the pleura, may reach the air-passages and appear as sputum; still more rarely, by communication of the esophagus or rupture of an aneurysm, particles of food or blood may pass this way. The secretion of the mucous membrane or of the glands of the throat, of the mouth, of the nose, and also other substances from these locations (as blood, micro-organisms, particles of food), mingled in various proportions with the expectoration, may give rise to error. Expectoration may be entirely wanting, even when the material for expectoration may be present in the air-passages in considerable quantity, when there is absence of cough, or when the cough is feeble;[1] finally, it may sometimes happen in all diseases of the respiratory organs that there is either no cough at all or only a dry cough. The blood evacuated from the stomach by vomiting may give occasion for swallowing, may then be expelled by coughing, and may thus be confounded with pulmonary hemorrhage; but, on the other hand, in hemorrhage of the lungs a part of the blood—sometimes a considerable quantity—may be swallowed and may give rise to symptoms of hematemesis.

When possible, it is best to collect the expectoration in a transparent glass vessel (as a matter of fact, we may readily understand that we shall generally have to employ a non-transparent receptacle). As much as possible mixture with other substances, as vomited matters, is to be avoided. A white porcelain plate, with one-half of its surface blackened with asphalt, enables one to scrutinize more exactly the expectoration. The expectoration upon both halves of the plate is to be examined, and, in order to separate it or to remove a portion for microscopical examination, we employ a pair of microscopic needles.

1. General Characteristics of the Expectoration.—We must take into consideration the *quantity*, *reaction*, *consistence*, or *form* (to which latter also belongs the quantity of air mingled with it and its arrangement in layers), its *color* and *transparency*, and, finally, its *odor*.

The *quantity of expectoration* changes with the amount of material which is in a condition to be thrown off, and this differs very much with different diseases, also with the strength of the cough. We have already referred several times to the influences that determine this. In general, patients with certain forms of *bronchitis* (broncho-blennorrhea) and with *cavities*, especially those with *bronchiectasis*, have the most abundant expectoration—a maximal amount of one or two liters a day. Sudden marked increase of expectoration occurs with the rupture of *empyema* into the lungs.

When not much contaminated with vomited matter the *reaction of the expectoration* is always *alkaline*.

From the above-mentioned general peculiarities (*consistence*, *form*, *color*, except only the *odor*) we may recognize, according to its chief constituents, in which class the expectoration belongs. Accordingly, we distinguish—mucous sputum, muco-purulent sputum, purulent sputum, serous sputum, bloody sputum.

Mucous Sputum.—This is either quite glassy and transparent or whitish-gray, generally with some consistence and tough; if more fluid, then it consists chiefly of saliva. It occurs in the first stage of *acute bronchitis* from the very slight—what may be called the physio-

[1] See p. 142.

logical—secretion of mucus in the trachea. Very often its source is higher up in the pharynx.

Muco-purulent Sputum.—This consists of a mixture of mucus and pus in varying proportions. The latter is recognized by its yellowish or yellowish-green color and its want of transparency. It may be distributed through the mucus in small particles or strings, or it may form larger flocks or balls held together by mucus; the latter, placed in water, are bullet-shaped; spread out upon the bottom of an empty glass, they sometimes flatten out in circular form (coin-shaped sputa in case of cavities, but sometimes also in ordinary purulent bronchitis, as in measles); finally, in the scanty spongy mucus with slight consistence, the pus of the separate sputa may run together ("confluent sputa"). If the sputum contains many air-bubbles, these cause the separate lumps and balls to float in the watery part of the sputum (serous fluid or very watery mucus or saliva). *Mucus in three layers* consists of an upper layer of masses and balls, which the air-bubbles cause to swim, and from which hang down into the second layer, slimy, purulent strings consisting of watery mucus and serum; on the bottom is a layer entirely confluent, like a deposit of decomposed pus (fetid bronchitis, gangrene of the lung).

Purulent sputum consists of almost pure pus, whose source is either an abscess of the lung which has given way or an empyema. Sometimes almost pure pus may be coughed up when there is a sudden very considerable discharge from a cavity. As it traverses the air-passages, there is always some mucus mixed with it.

Serous sputum is a special peculiarity of the sputum of *edema of the lungs.* It is very fluid, although, on account of the admixture of mucus, not so much so as blood-serum, being mixed with mucus. It consists of blood-serum, and hence contains albumin; for this reason it retains air-vesicles for a long time, as do all fluids containing much albumin; it is markedly frothy. It is either a quite light gray and transparent, or, as is frequently the case, like beef-juice, owing to a slight admixture with blood; when containing much blood it is the color of prune-juice (edema of the lungs with pneumonia).

Bloody Sputum.—All of the varieties of sputum previously mentioned may be mixed with blood. Slight mixture of blood is seen in the expectoration of tough mucus as bloody streaks. These are generally mixed from the upper air-passages, often from the throat or nose, yet sometimes they arise from the lungs or the smallest bronchial tubes, as in pneumonia. A small quantity of blood, with partial escape of coloring matter of the blood, intimately mixed with tough, glassy mucus, colors the sputum uniformly bright red with a greenish tinge, or, by transformation of the coloring matter of the blood, makes it yellowish-red, rusty, even greenish (all of these with *pneumonia*). In muco-purulent sputum blood appears either in streaks or as little spots, as in phthisis, or intimately mixed: the pus is then reddish-yellow, brownish-yellow, or more markedly reddened, as it more particularly occurs in bronchiectasis and phthisical cavities, but also sometimes in atypical lobar and broncho-pneumonias. Brownish bloody sputum in phthisical subjects may always be referred to a complicating broncho-pneumonia. When there is only a small admixture

of blood serous sputum, as was mentioned above, is the color of beef-juice.

If there is *considerable hemorrhage* with the expectoration, it is markedly colored with blood; sometimes there may apparently be no sputum, but fluid blood may be expectorated in a liquid state, coagulating afterward, This is described as *hemoptysis.* When a pulmonary hemorrhage is quickly coughed up, the blood is bright red and frothy from being mixed with the sputum, but sometimes it gushes out in such quantity that there is no cough. It is distinguished from blood that comes from the stomach, in that the latter, from longer stagnation and from the effect of the secretion of the stomach, generally is darker, quite brown, like coffee-grounds; besides which it is often mixed with food and *has an acid reaction.* Yet the blood from the lungs, though only when there is considerable quantity, may be also dark, even black-red, if it has stagnated in the lungs or air-passages: thus, a patient who has had an hemoptysis may continue for a whole day to throw off markedly bloody sputum, which becomes more and more dark in color.

Hemorrhage of the lungs occurs very much more frequently with tuberculosis than from other causes. In this disease there occur all varieties of hemorrhage, from the scarcely visible particles of blood or a slight coloring of the purulent discharge from a cavity to the profuse, almost immediately fatal hemorrhage. Moreover, in *infarction* of the lungs there may be bloody sputum, or even pure blood may be discharged. *Croupous pneumonia* and *edema of the lungs* are generally accompanied with slight quantities of blood intimately mixed with the sputum.

Sometimes it is perfectly easy to diagnose hemorrhage of the lungs, and again it is extremely difficult. Particles and streaks of blood occurring in the midst of purulent material are very suspicious. If they occur with gray mucus, it is generally quite unimportant, because in the latter case they usually come from the pharynx or its neighborhood; but when there is considerable hemorrhage there may be doubt as to whether the blood comes from the stomach or lungs if the blood is expectorated very rapidly, and so is yet bright red, and if, during the act of vomiting, some blood is aspirated and causes cough. On the other hand, blood from the lungs may seem to come from the stomach if, from stagnation, it is unusually dark or if a part of it is swallowed and then vomited. When the patient is unconscious or asleep, blood from the nose or throat may be drawn into the air-passages, and then, after considerable has accumulated, be coughed up, but more frequently it flows into the stomach. In the latter case, by inspection of the throat we may sometimes see a streak of blood marking the track upon the posterior wall of the pharynx. In all such cases a decision is to be reached by the most careful examination of the lungs, stomach, and nose.

A peculiar sputum, like raspberry jelly, is observed in cases of *tumor of the lungs.*

Sometimes in *hysteria* there is an expectoration from the pharynx or esophagus of a peculiar raspberry red, which may mislead one (years ago described by E. Wagner).

Green Expectoration.—When sputum is left to stand, say in a jar, for some time, it becomes green. We now know, from the investigations of Frick, that this is due to a green-producing bacillus. This change of color is without any diagnostic significance. Regarding sputa which are green when expectorated compare the next following pages.

The odor of sputum is ordinarily stale; when it is scanty, it is often offensive from mixture with the secretions of the mouth, especially among the lower classes or when the patient is very sick. Purulent sputum from a cavity, if it has been long retained, may be putrid or have a peculiar putrid-rancid odor (only with phthisical patients *in extremis*). In cases of *fetid bronchitis*, *bronchiectasis*, and *gangrene of the lungs* a more marked and very characteristic, sharper and more penetrating, quite offensive odor from the muco-purulent sputum decomposing in the air-passages is commonly present; but in the last-mentioned disease it may be entirely wanting (*"odorless gangrene"*). Offensive odor of sputum may sometimes be caused by decomposition of particles of food in the mouth or by offensive plugs in the lacunæ of the tonsils, and thus one may be entirely deceived.

A penetrating, aromatic, fruit-like odor of sputum, resembling the odor of stewed prunes, has lately been described by Eichhorst. It precedes the rupture of an echinococcus into the air-passages and the appearance of the membranes in the expectoration.

2. Foreign Substances in the Sputum which are Visible to the Unaided Eye.—Nowadays the microscopical examination of the expectoration with its brilliant, but partial, results, is carried to such an extent, and so calls the chief attention to this secretion, that it seems necessary to draw attention to the importance of examining it with the unaided eye. Carefully conducted, it not infrequently brings the physician, in difficult cases, directly to a correct diagnosis, besides facilitating the use of the microscope in showing how to find the right spots from which to take the particles for closer examination.

The inhalation of *coal-soot* (most frequently by those especially exposed to it, but also by all dwellers in cities) colors the sputum, in streaks or diffusely, blackish-gray. When *iron-dust* is inhaled, it colors the sputum quite black or ochre-yellow and red.[1] When the sputum is scanty it is more deeply colored than when it is abundant, since in the former case the coloring-matter is more concentrated.

We have already referred to the addition of blood. The presence of *hematoidin* is sometimes evident to the unaided eye by a yellowish or brownish-red color in separate spots; it occurs in the lungs when there is *disease of the heart*, in cases of *abscess of the lungs*, and in *empyema*.[2]

In icterus the *bile-pigment* is sometimes present in the expectoration; I have often observed that in *pneumonia with icterus*, more particularly, it colors the sputum a distinct yellow-green or green.[3]

In *abscess of the lung* we observe lung-tissue in the shape of larger or smaller pieces. These "lung sequestra" may sometimes be very

[1] See on this point, also, under Microscopical Examination.
[2] Confirmation by the Microscope, see below.
[3] Compare also what has been said above about green coloration of sputum that has been standing some time.

large—2.5 cm. long (Salkowski of Leyden). Pieces of cartilage from the trachea or the bronchial tubes in deep *ulceration* and the accompanying *perichondritis* of these organs will sometimes be coughed up.

Fibrinous tubes, formed in the bronchial tubes as a result of fibrinous inflammation there, may form a more or less considerable part of the expectoration. We may have a firm cast of an entire dichotomous



FIG. 35.—Large bronchial coagulum (chronic fibrinous bronchitis) (after Riegel).

ramification of a large bronchial trunk, even to the finest branches (even to the alveolar tubes and the alveoli?); more frequently they come from the smaller bronchi, and are only divided two to five times. Very often these casts are thrown off while they are fresh, as is evident by their white color; they are also often yellowish-brown, or else reddish, from the addition of blood. They are often found as irregular

lumps covered with mucus or small flakes, so that the inexperienced do not recognize their true character. In order to make them out it is necessary to isolate them by shaking them up with water in a test-tube.

Generally they exist only as casts of the smaller bronchial tubes in *croupous pneumonia*, and are most abundant shortly before and during resolution, as dense large casts in *chronic croupous bronchitis*, and in *acute croupous bronchitis* in consequence of laryngeal and tracheal croup.

Complete casts of the trachea, and even of the larynx, are sometimes thrown off in croup. Casts wholly from the smallest bronchial tubes, or, in reality, from the alveolar channels, occur in bronchial asthma, and more rarely in croupous pneumonia, as the so-called *spirals*. If they are small, they form in the expectoration (compare page 154) diminutive gray transparent or whitish opaque flocks or lumps which frequently, on close examination, look like fine hairs rolled together. The finest forms of these spirals, the so-called nude central threads, are most frequently found in light gray, egg-shaped or globular corpuscles scarcely the size of a millet-seed.

Regarding echinococcus bladders and the exotic *Distoma pulmonum* (Bälz) found in the sputum, see under Microscopical Examination, page 157.

Of the *crystals* occurring in the sputum (which, of course, can only be perfectly made out by examination with the microscope), sometimes by careful examination with the naked eye two forms may possibly be recognized. In the fetid sputum in three layers (fetid bronchitis and gangrene of the lungs) there exist peculiar grayish-yellow, very offensive lumps, which may be barely visible or may be as large or larger than lentils; these lumps enclose fat-crystals.[1] These same bodies occur as offensive plugs from the lacunæ of the tonsils, although never in so large a quantity as in the other conditions. Hence when they are found in the sputum we must always carefully examine the tonsils.



FIG. 36.—Bronchial coagulum, natural size, with croupous pneumonia.

In this disease the small forms are very frequent, the large ones very rare, but frequent with chronic fibrinous bronchitis.

Further, in chronic *croupous bronchitis* and in *bronchial asthma* there are found imbedded in the sputum, sometimes adhering to the concretions, peculiar small bodies, yellowish kernels, like grains of sand, which easily strike the practised eye; these, generally numerous, are the so-called Charcot-Leyden's crystals.[2]

It remains to mention some fungi found in the sputum whose presence may be indicated by the macroscopical examination, but this examination would be without diagnostic value unless confirmed by the microscope. Different kinds of mould, especially *Aspergillus*

[1] See further, p. 156.

[2] See p. 156*f*.

fumigatus, are very rarely found, except as a pathological result, and generally in phthisical and bronchiectatic cavities, which are noticed as gray or greenish little collections; *muguet*,[1] as white tufts almost always arising from the mouth and throat (hence, these are to be carefully examined); only in quite isolated cases they come from the upper air-passages.

The finding of *actinomyces* in the expectoration is of greater importance, but of yet greater rarity. It can be recognized by the naked eye by the little kernels of uniform size, shaped like millet-seeds, greenish-yellow or yellowish-white, sometimes somewhat glassy (I have seen them in one case, since then they have been seen several times by others); of course they are only to be accurately recognized by the microscope.

There is another fungus, occurring in granules much like those of the actinomyces, which can scarcely be mistaken for anything else, the so-called *leptothrix buccalis*. The granules are smaller, more irregular, and more whitish than those of the actinomyces—sometimes like little scales, sometimes not distinguishable from minute bread-crumbs. Under the microscope they consist only of fungus threads, which are arranged after the manner of the leptothrix, and give the same reaction.[2] Such sputa sometimes (not often) on standing gradually become yellowish or develop a yellow coating—luxuriated leptothrix, as has been hitherto assumed. The granules are particularly found in chronic bronchitis and bronchiectasis. The finding of large masses of *tubercle bacilli* is sometimes made easier by the presence of yellowish, generally flat lumps—"lentils"—in the sputum from cavities, which, besides, usually contains many elastic fibers[3] and, also, although much more rare, if there are small white (barely visible) scales, very much like those of which the artificial pure culture of the *bacillus tuberculosis* consists. Both elements, especially the latter, usually contain or consist of masses of bacilli. It is very easy to be deceived by the admixture of food-particles in the sputum. Chiefly is this the case from minute bread-crumbs and small white lumps of coagulated milk (which not infrequently contain fat-crystals).

3. Microscopical Examination of the Sputum.—Small particles are placed under a glass cover, which is to be only moderately pressed. It is to be examined with a No. 7 or 8 Hartnack, or DD or F Zeiss.

In all mucous and muco-purulent sputum there are *threads of mucus* and *mucous corpuscles;* the former are more sharply defined the tougher the mucus is. In pneumonia and asthma they are often spiral, and in these diseases they pass by imperceptible gradations over into the finest and most delicate fibrinous formations.[4]

White blood-corpuscles are found in all expectoration, but in much greater numbers in the purulent parts. They are generally of various sizes, granular, not infrequently filled with drops of fat and myelin or contain particles of soot; or also, in rare cases, contain minute lumps of hematoidin.[5] Regarding the occurrence of greater numbers of eosinophile cells in the sputum of asthma, see page 157.

[1] See this.
[2] See below, p. 158.
[3] See below, p. 152.
[4] See Spirals, p. 154.
[5] See p. 155*f*.

Red blood-corpuscles are found in the different kinds of bloody sputum, generally with the form well preserved, but often paler, even as rings; when the sputum has been retained for a long time they are granular.

Epithelium.—Flat epithelial cells from the mouth are a common ingredient of the sputum. They are easily recognized by their size and thinness, the latter manifests itself by numerous cracks and folds. Flat epithelium, which probably comes from the esophagus, occurs in large clusters in the so-called bloody sputum of hysteria.

Changed *cylindrical epithelium* of the air-passages in the form of mucous and goblet cells are observed in all cases of catarrh of the trachea or of the bronchi, and sometimes in large numbers. On the other hand, it is rare to find these epithelial cells in their original condition, with homogeneous protoplasm, with bladder-like nucleus, covered with cilia; and still more rare to obtain the motion of the cilia or to find it responsive to heat. The possible origin of these cells in the nose is not to be overlooked. They have diagnostic value.

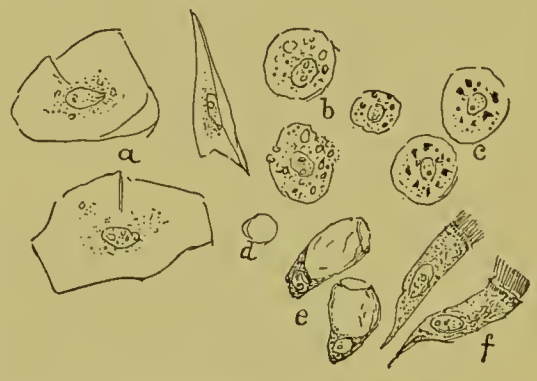


FIG. 37.—Epithelium from the sputum: *a*, flat epithelium from the mouth; *b*, the so-called alveolar epithelium, containing little drops of fat and myelin; *d*, a red blood-corpuscle; *e*, mucus-cells; *f*, oscillating cells.

The so-called *alveolar epithelium* (see Fig. 37) was formerly considered an important constituent of the sputum. But it is neither possible to affirm its source nor to give its diagnostic value. There are elliptic or round, not infrequently somewhat flattened, cells with an often indistinguishable nucleus (made visible by the addition of acetic acid), larger than the ordinary white blood-corpuscle. The protoplasm is fine or coarsely granular, sometimes filled with drops of fat or myelin (Virchow); also we may see complete fatty degeneration with formation of large fat- and myelin-drops. These cells contain particles of coal- or iron-dust (the latter made dark green by sulphide of ammonium, blue by yellow prussiate of potash and muriatic acid). This alveolar epithelium occurs in bronchitis and all kinds of acute and chronic *pneumonia*, hence does not have any diagnostic value. Its epithelial character is not at all constant. I think it quite probable that it is mostly or altogether made up of white blood-corpuscles, enlarged by metamorphosis of their protoplasm, and partly by absorption of small particles. In part, also, this may come from the deeper layer of the bronchial epithelium (Panizza, Fischl, Senator).

The so-called *heart-disease cells* are cells which show great conformity with those last mentioned in shape, size, form, and visibility of their nucleus, but they are partly filled with very fine, partly with larger, yellow or brownish little nucleoli. This nucleolus consists of hemosiderin, an iron-containing derivative of hemoglobin (F. A. Hoffmann). These cells are no doubt principally leukocytes, a small number of them also being alveolar epithelia. If they are present in con-

siderable quantity, they give a yellowish-brown tint to the sputum or to parts of it. These cells are almost pathognomonic of *conditions of the lungs associated with heart-disease—i. e.* brown induration of the

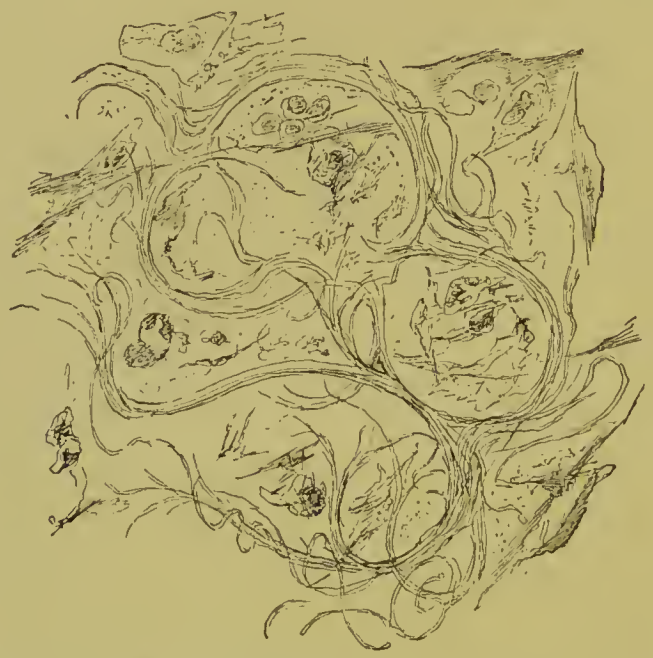


FIG. 38.—Elastic fibers, from a mass in the sputum from a phthisical cavity, without change. Zeiss, F. Oc. 3.

lungs, which is caused by long-continued defective flow of blood in the lesser circulation; disease of the mitral valve, myocarditis, adhesive pericarditis. Similar cells are also seen in small numbers in cases where blood appears in the sputum, particularly where there is old bleeding [extravasations], as infarction of the lungs, slightly bleeding bronchiectasis, also pneumonia.

Elastic fibers are an important constituent of sputum, since they infallibly show the destruction of lung-tissue (less frequently of the tissue of the bronchi), but still more because they indicate a severe disease of the lungs often before there are physical signs of it. They occur in *tuberculosis, gangrene, abscess of the lungs.* They generally have a double outline; now and then there are branching fibers which have a serpentine course or large irregular curve. They generally lie in bundles, and often show the structure of the lung-vesicles. (Compare Figs. 38 and 39.)



FIG. 39.—Elastic fibers, sedimented after treatment of sputum with alkali.

They always exist in clusters and with a remarkably alveolar arrangement in the shreds of lung-tissue in abscess of the lungs and

when there is suppurating gangrene; further, almost always in the so-called "lintels" of tubercular sputum. When elastic threads occur singly, which may be in all the conditions named, it is very difficult to say which is their special cause. Then, also, it is not easy to distinguish them from fat-crystals,[1] and farther from elastic fibers in food. Besides, since the discovery of the bacillus tuberculosis their importance for the early diagnosis of phthisis has disappeared; but for determining whether we have a more or less destructive form of phthisis they are as valuable as ever.

Method of Examination.—A suspicious particle of sputum is placed on the slide, then there is added either pure water or a drop or two of a 10 per cent. solution of potassium hydrate, and the cover-glass is put on. In the solution of potassium hydrate all particles of tissue swell to a uniform jelly, except the elastic fibers, which stand out distinctly.

To obtain elastic fibers when they are not present in quantity, a portion of sputum is boiled with an equal quantity of an 8 to 10 per



FIG. 40.—Curschmann's spirals, natural size (after Curschmann).

cent. solution of potassium hydrate; then the jelly-like mass is to be diluted with water and allowed to stand for twenty-four hours. The elastic fibers, as distinct organic substances, settle to the bottom, but are often much swollen and not readily distinguished from fibers of the food. The elastic fibers may also be separated in a few minutes by the centrifuge.

In individual cases of *gangrene of the lungs*, but by no means in all, elastic fibers may be wanting; possibly they may be destroyed by the action of a ferment (Traube) [see page 164]. Moreover, simple gangrene of the lungs is rare; we generally have a suppurating gangrene, and this can hardly fail to furnish the shreds of lung-tissue previously described.

[1] See under, p. 156.

Spirals (Leyden, Curschmann, and others).—By this name we understand spiral forms which are found almost exclusively in the coherent mucous sputum of bronchial asthma, but are also found in other sputa having the same ropy, mucous consistence.

FIG. 41. FIG. 42. FIG. 43.



FIGS. 41–43.—Curschmann's spirals: *a*, central fiber (after Curschmann).

With a little practice the larger of these spirals can be recognized by the naked eye. If placed under a cover-glass and slight pressure is made, so as to spread the sputum out thin, even without a magnifier spiral fibers can be seen, and not infrequently in their interior a shining stripe, which generally is wavy. It is easy to imagine that this stripe is a long-stretched spiral. If slightly magnified by a good magnifying-glass or a Zeiss AA with ocular No. 2, it can be distinctly seen that in its outer parts the spiral is formed of loose, corkscrew-like fibers, while the shining, bluish-shimmering stripe in the interior appears more homogeneous. But if still more strongly magnified, we recognize on the stripe also a few very fine spiral threads. The loosely-wound exterior part of the spiral has lately been frequently called the mantle spiral,



FIG. 44.—Nude central threads in asthma sputum; mag. 240×.

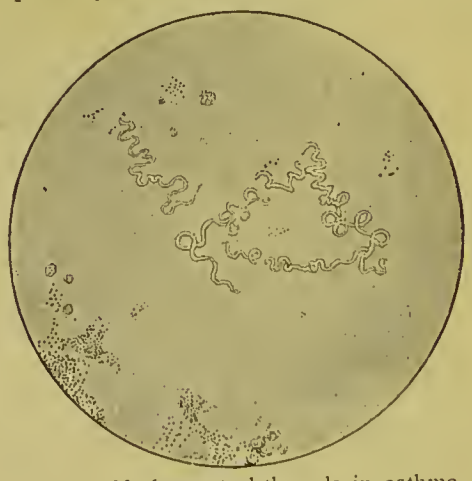


FIG. 45.—Nude central threads in asthma sputum; mag. 585×.

while the central stripe, as proposed by Curschmann, is called the central fiber.

In the mantle part of the spirals cells are always present: very frequently numerous round-cells containing one nucleus, with eosinophi-

lous granulations, sometimes of considerable size. Also asthma-crystals may be found here, and occasionally cells containing hemosiderin.

Often the spirals, particularly the larger ones, are without the central thread. On the other hand, if strongly magnified, the central threads are found in the midst of small balls of sputum without mantle spirals—isolated or nude central threads. The finest glassy little flocks of asthma-sputum, described above,[1] very often contain a good many spirals, and some of them are quite long, although very fine (Figs. 44 and 45). By the Weigert fibrin stain, which also stains the mucin, Schmidt has demonstrated them in hardened sputum. With some practice they are easily found without staining.

According to the color-reactions, there can be scarcely any doubt that the central threads consist of mucin. They are certainly not the artificial expression of a cavity, as was formerly frequently supposed. It may further be supposed that the central threads do not signify anything more than that the innermost part of the spiral has been consolidated and afterward stretched.

These formations are found in bronchial asthma to such a prevailing degree that they are of important significance for the diagnosis of this disease. They are usually most abundant in the expectoration which is thrown off toward the end of the attack. Possibly also they participate in producing the attack. They are also found in small numbers in quite different states, especially in chronic, obstinate bronchitis and emphysematous bronchitis, in bronchitis accompanying heart-disease, in croupous pneumonia, and here, usually simultaneously with coagulations, also in very chronic pulmonary tuberculosis, and finally in hemorrhagic infarction.

The conditions in which they appear all have this in common: a coherent mucous sputum and a certain degree of dyspnea. It is remarkable that where there are the most ropy sputum and the severest dyspnea, as in bronchial asthma, the spirals are most numerous and most perfect.

Gerlach by twisting particles of ropy mucus has produced not only spirals, but also central thread-like formations; but it has not yet been proved that these artificial central threads have the same strong affinity for certain anilin colors as the natural ones have. The suggestion naturally arises that in the bronchi also these forms originate by the twisting of the particles of sputum. This seems to be self-evident at once if we consider the appearance of the spirals. But how and where this twisting takes place no one has yet shown. Schmidt found spirals with central threads in the upper lobes of a diseased asthmatic patient in the majority of the bronchi that had a diameter of one to three millimeters. This, thus far, is the only fact which directly bears upon the question; everything else bearing upon the place and manner of formation of the spirals is merely conjecture.

Starch-corpuscles.—These are often found in *hemorrhage of the lungs* (Friedreich), and in *gangrene* (von Jaksch), but are as yet without significance.

Crystals.—*Crystals of hematoidin* are brownish-yellow if pure, of a shining color, rhombic plates or fine needles, and these single, or two

[1] See p. 149.

or three crossed, or in tufts. The crystalline formation may also occur as grains and lumps; not infrequently in the center is a white blood-corpuscle, and it may be that the needles are arranged with their points



FIG. 46.—Crystals of hematoidin.



FIG. 47.—Needles of fatty acids (after Strümpell).

standing out from the cells. They indicate that blood has been long retained: in *gangrene* with formation of abscess; in the *pus of empyema* which has perforated a long time before, as in one case that came under my observation of a slow hemorrhage into the lungs from a thoracic aortic aneurysm. Sometimes there are spots macroscopically visible when there is hematoidin in the sputum.[1]

Crystals of fatty acid (margaric-acid crystals, see Fig. 47). They are long, thin, slender needles, slightly or very markedly bent, which are found singly, in large bundles or nodules, or quite irregularly arranged. They are generally distinguished from elastic fibers by the uniformity of their curving. When a portion of sputum is dried in the air without heat, they are completely dissolved upon the addition of ether, while the elastic fibers under the same circumstances are not changed. They occur generally in masses in *gangrene of the lungs* and *fetid bronchitis*, and especially in the lumps or plugs previously mentioned;[2] they are also found in the plugs which are formed in inflamed tonsils;[3] finally, they may occur singly in any muco-purulent sputum, especially after standing in a warm place for some time.

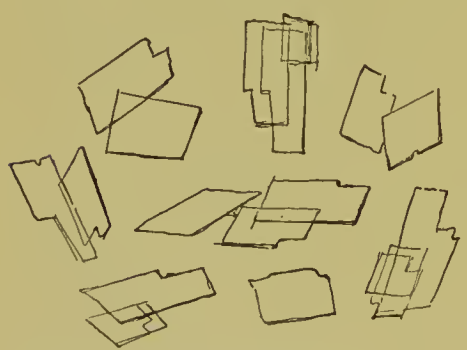


FIG. 48.—Crystals of cholesterin (after Strümpell).

Cholesterin Crystals.—These are thin rhombic plates with the corners cut out, which become green and then red when treated with dilute sulphuric acid and tincture of iodin. They are sometimes found in old perforating pleural pus, also in tuberculosis.

Charcot-Leyden's Crystals.—These are slight, somewhat blue, shining, elongated octahedrals of great variety of size, sometimes visible

[1] See p. 146. [2] See p. 149. [3] See p. 149.

with a simple microscope, often only to be seen with a No. 8 Hartnack. They seem to be identical with the crystals found in the blood and marrow in *leukemia*, also sometimes occurring in the feces.[1] They probably consist of a mucous substance (Salkowski).

As a sign of *bronchial asthma* they are of great diagnostic importance;[2] they then occur most abundantly during and after the attacks (Leyden). They are less frequently found in *acute bronchitis, chronic croupous bronchitis,* and *tuberculosis.*



FIG. 49.—Charcot-Leyden's asthma-crystals (after Riegel).

In the expectoration of asthma the points where these asthma-crystals are found can often be easily recognized with the naked eye as dry crumbs.[3] They are often mixed with peculiar, fine, granulated round-cells which look as if filled with dust; at the same time with these are found spindle-formed figures with a slight glistening—a transition stage to Charcot's crystals (?). F. Müller and Gollasch lately found that the granulated cells are eosinophilous.[4] The spindle-shaped formations also contain eosinophilous granules. These crystals and eoscophilous forms are found especially numerous upon and in the "spirals."

In isolated cases there are found in the sputum *tyrosin* (*fetid bronchitis, empyema*, according to Leyden), *oxalate of lime* (*diabetes*, Fürbringer; *asthma*, Ungar), and *triple phosphate*.[5]

Animal Parasites.—We may have whole echinococcus bladders or their fragments (recognized upon cross-section by the remarkably uniform streaking (see Fig. 51), and also the hooks of the scolices (Fig. 50) in the sputum in case one of these parasites enters the bronchial tubes by rupture from the lungs or liver. (They are slightly magnified.)

[1] See under Stools. [2] See Spirals, p. 154. [3] See p. 149.
[4] Compare the Blood in Leukemia.
[5] See chapter on Urine, for illustrations of these substances.

The *Distoma pulmonale* (Bälz), which causes hemorrhage without any other manifestation, declares itself by its eggs in the sputum (to be seen by the simple microscope). It is found in foreign countries, especially Japan.



FIG. 50.—Echinococcus (scolices) (hooks, after Heller).

Infusoria (*Monas, Cercomonas—Kannenburg*) are found in gangrene; they are seemingly without pathological significance.

Fungi:[1] *Leptothrix buccalis*, as has already been mentioned, page 150, is present in the yellow scum arising on sputum that has been standing some time, besides the fatty-acid crystals in the bronchial plugs in putrid bronchitis, and also occurring separately in granules which resemble those of actinomyces. Either it is first mixed in the sputum in the mouth or it has entered the air-passages from the mouth, but it is present there without any known pathological significance. *Specific reaction:* With iodin and potass. iod. it is stained blue-red. [For formula, see page 163.] Without this reaction it may be confounded with elastic threads, even with fatty acids.[2]

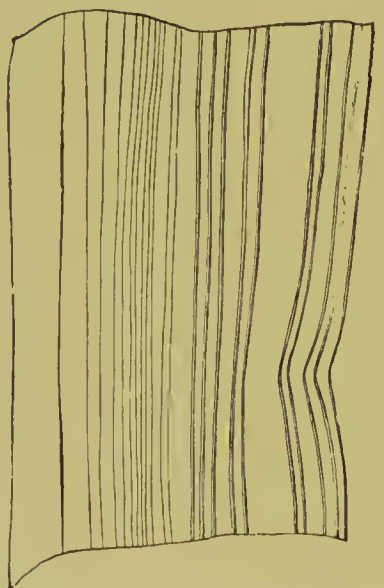


FIG. 51.—Echinococcus membrane, cross-section enlarged.

[1] For the macroscopical evidence of the presence of some of them, see page 149*f*.

[2] See the chapter on the Digestive Apparatus and Microscopical Examination of the Contents of the Mouth-cavity.

Sarcina pulmonalis is a fungus formed by division from developing endogenous spores (Hauser). While similar, although smaller, it has nothing to do with sarcina ventriculi. The recent views upon its frequent presence may be somewhat questioned (confounded with *Micrococcus tetragenus* (?), Flügge). It has no known pathological significance.

Tubercle Bacillus (Koch).—This generally occurs in the purulent parts of the sputum of *tuberculosis* of the lungs or trachea. Exceptionally it may be mixed with the sputum from the throat or nose if a tubercle breaks up at that point. The bacilli are generally very abundant in the so-called "lintels," and in rare cases as small pure cultures in the tiny white scabs which were spoken of above, page 150. These split fungi are straight or moderately—rarely much—bent, very thin rods of somewhat variable length, 2 to almost 4 μ[1]—that is, about the diam-



FIG. 52.—Tubercle bacilli in the sputum, first colored with anilin-fuchsin, and then with methylen-blue. Zeiss's homog. immersion $\frac{1}{12}$, Oc. 4, camera lucida drawing; magnified about 1000 diam.

eter of a moderate-sized white blood-corpuscle. They often contain spores. On account of their thinness, and because they are without motion, they are with difficulty seen in the sputum unless they are colored. In order to bring them into view we stain them, and by a method which at the same time produces a special reaction, a very certain proof that it is the tubercle bacillus and not one of the numerous other bacilli. It is to be magnified 600–400, or, for those accustomed to examine for it, 300, diameters—that is to say, with a $\frac{1}{12}$ oil immersion lens (with an Abbé condenser), or a Hartnack No. 8 or at least No. 7, Zeiss F.

Microscopical Demonstration of the Tubercle Bacillus.—Only three

[1] [The Greek letter μ represents one-thousandth of a millimeter ($\mu = 0.001$ mm.), and is the sign of a *micro-millimeter* or a *micron*.]

of the numerous methods of staining are mentioned here, the second of which—Ziehl-Neelsen—is the one most in use at the present time.

I. (Weigert, Ehrlich).—With perfectly clean needles we place some sputum upon a plate with a black surface, and there spread it out with the needles. From this is selected a suitable portion,[1] which is placed upon a glass cover, and then it is to be broken up with the needles. Upon this is now placed another glass cover, and the two are pressed firmly together. What is squeezed out upon the edges is to be washed away, and then the two glasses are to be carefully separated, so that there may remain upon each the thinnest possible layer, equally distributed. These are then laid aside to dry. Then 12 drops of anilin oil are thoroughly mixed with a small test-tube full of distilled water, it being shaken till it is intimately mixed. The mixture is allowed to stand for a short time, and then some of it is to be filtered through a moistened filter into a watch-glass. From a previously prepared concentrated alcoholic solution of fuchsin there is then to be added sufficient to make the mixture opaque or to cause a slight metallic shimmer to appear upon the surface; about 6 drops are necessary. Good fuchsin S. is necessary.

The glass covers are allowed to dry in the air, and then each is passed three times through the flame of a spirit-lamp and laid in the coloring solution with the sputum side down. The watch-glass, covered over, is allowed to stand for twenty-four hours, or it is slowly warmed over the spirit-lamp until a slight deposit of moisture appears not only upon the edges, but also upon the middle, and then it is set aside for about ten minutes.

The manipulation is continued by washing the glass cover in water, and then for a few seconds dipping it in a mixture of 1 part of nitric acid and 2 of water (without letting go of it with the pincers) until it, being again washed in water, continues to show a slight red shimmer. Then the preparation may be immediately examined in water: the tubercle bacilli are colored an intense red, while all the rest is colored a pale reddish tone. It is advisable to stain the glass cover a second time with a watery solution of methylen-blue. This is done by placing it in this solution for a minute or two after taking it out of the acid mixture and thoroughly washing it with water. Then it is again washed, when it may be examined.

Instead of fuchsin and methylen-blue, we may, in exactly the same way, employ gentian-violet and Bismarck-brown. The preparations are preserved by first drying them in the air, then passing them three times through the flame before laying them upon slides upon which has been placed a drop of xylol-Canada balsam.

The decolorizing with the nitric-acid solution must not be too prolonged, else the bacilli lose their coloring. With preparations that are to be preserved the nitric acid must be very carefully removed by repeated washings with water, because the acid destroys the color.

The alcoholic gentian-violet, as well as the fuchsin solution, retains its color very well. Sometimes the Bismarck-brown, and also the methylen-blue, must be filtered before using. Besides these, one needs for his work a black plate, two quite long microscopical needles which

[1] See above.

must be heated red hot each time before using, a pincette with broad beak, some watch-glasses, glass slides and covers, and a spirit-lamp.

II. (Ziehl-Neelsen).—In this method, instead of the anilin-water fuchsin, there is employed a mixture of 90 parts of a 5 per cent. solution of carbolic acid and 10 parts of concentrated alcoholic solution of fuchsin. Staining is also done by warming, and in everything else the same as in I.

III. (Gabett).—A briefer but less certain, and hence less useful, method has been recommended by Gabett: A dry preparation which has been passed through a flame is placed for two minutes in a solution of 1 part of fuchsin S. in 100 parts of a 5 per cent. solution of carbolic acid and 10 parts of absolute alcohol, and then, immediately after, for one minute in a solution of 2 parts of methylen-blue to 100 parts of 25 per cent. sulphuric acid. It is rinsed with water, and then, for preservation, is dried and mounted in Canada balsam. The preparations, if successful, are very beautiful and permanent; but the discoloration is difficult to control, because it is carried on in the colored solution: sometimes it is too strong, sometimes too weak. It is best not to attach any importance to a negative result.

Note.—For handling the cover-glass it is best to use a Cornet's pincers. With some practice the staining solution may simply be dropped on the cover-glass, and then this is heated directly over the flame till it steams, but is not allowed to boil. The staining solution is added drop by drop on the glass, so as to keep it moist. If by chance there are thick portions in the preparation which are difficult to decolorize, it may be dipped alternately into a 30 per cent. solution of acid and into alcohol in order to decolorize the preparation.

For demonstrating the bacilli when they are scant in numbers we can very strongly recommend Biedert's method: A tablespoonful of sputum and two of water are mixed with four to eight drops of solution of soda (according to the condition of the sputum), and then boiled in a saucer, constantly stirring and gradually adding from four to six tablespoonfuls of water. The boiling is continued till a homogeneous fluid is formed. This is then centrifugated energetically or allowed to stand for two days (not longer) in a conical glass. Bacilli (and also elastic fibers) form a sediment. Then it is decanted, and a sample is removed with a perfectly clean instrument which is treated like a sample of sputum. In order to have it adhere it is sometimes necessary to add to each cover-glass a particle of untreated sputum of the same patient. Staining is done after the Ziehl-Neelsen method.

Recently Dahmen has proposed to evaporate the sputum in a water-bath at 100° C. [212° F.], then triturate the sediment in an agate mortar and treat it on a cover-glass. The procedure is impractical, and is less certain than the preceding method.

When one is not accustomed to examine for tubercle bacillus for the purpose of controlling the degree of staining, he should at the same time stain some sputum that is known to contain the bacillus [or he should keep test-slides on hand].

By these methods the *tubercle bacilli* are distinctly recognized by their red (or blue) staining. Since the spores that may be present are not stained, they may be seen in the interior of bacilli as clear points, and they may be so abundant as to cause the bacilli, when only slightly magnified, to look like the chain coccus (Fig. 52).

The direct culture of tubercle bacillus, as has been accomplished by Kitasato, has no diagnostic significance.

The number of bacilli found in a preparation depends, at least by the ordinary methods, very much upon chance, since only one or more particles of sputum at discretion are examined. Biedert's procedure rather permits one to draw some conclusion as regards the actual abundance of the bacilli in the lungs, but at best only an approximate estimate can be made in this manner as to the severity of the disease.

The presence of this bacillus in the sputum indicates *tuberculosis of the lungs* (unless there may be tuberculosis of the larynx), and its diagnostic value is so much the greater in that bacilli may often be discovered when the physical signs are indistinct or are altogether wanting.

Absence of the bacilli at a single examination is without value, especially when the sputum is scanty and not purely purulent. If they are absent in repeated examinations, this fact is to be considered with greater caution. On the other hand, in sputum that is not too scantily purulent the constant failure to find bacilli points with greater probability against tuberculosis. It is to be understood that the staining material is as it should be,[1] that the staining has been properly done, and that the most careful examination of the preparation has been made. In doubtful cases we recommend Biedert's sediment-process, described above, which gives tolerably certain success in finding bacilli when they are not numerous.

Koch's tuberculin, of which we will speak again farther on, has, among others, certain effects upon the sputum of tuberculosis of the lungs, which effects are sometimes of diagnostic significance. In the first place, the sputum becomes more abundant, usually more rich in bacilli; a sputum containing bacilli appears where before there was no sputum at all or no sputum containing bacilli. Besides, the bacilli very frequently show in a very decided manner the resemblance to chain cocci, mentioned above, and at the same time they frequently seem sharply bent, fallen to pieces, with the fragments lying together in irregular heaps. We here pass over these, and also the phenomena of Koch's reaction, since we refer to it again on page 164*f*.

Pneumonia Cocci.—(*a*) *A. Fränkel's Pneumococcus.*—These cocci appear in the sputum mostly in distinct capsules, and preponderantly, but not exclusively, as diplococci. They are oval or lance-shaped by becoming slightly narrower on the ends, which are turned in opposite directions. For the other characteristics compare Fig. 53. They may be stained by all anilin dyes, and are not decolorized by Gram's method. (*b*) *Friedländer's pneumococcus.* It is very similar to the former, but it is decolorized by Gram's method. (*c*) *Pio Foà's coccus*, which is a diplococcus enclosed in a capsule. So far as we know, it has only been found in the tissue-juice of pneumonic lungs, and has not been demonstrated in the sputum. It has great similarity to Fränkel's coccus, both by its lancet form and because it can be stained by Gram's method.

Fränkel's coccus is found in the lungs and also in the sputum in a majority of cases of pneumonia. It is scarcely to be doubted that it is an exciting cause of pneumonia, not only of the genuine croupous, but

[1] See above.

also of certain secondary, forms. It has likewise been found in the pus of empyema and of cases of meningitis which complicated a croupous pneumonia. But it is sometimes also found without any connection with pneumonia in pleuritis, metastatic meningitis, otitis, arthritis, in phlegmons, in peritonitis with perforation, etc. This coccus seems, therefore, to produce not only croupous pneumonia and its metastatic diseases, but also other forms of pneumonia and independent inflammations in a number of most different organs. It is a widespread, but frequently not very malign, exciter of inflammation.[1]

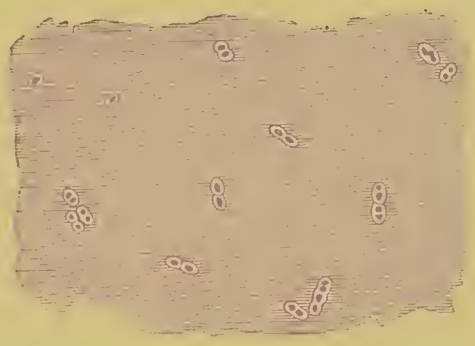


FIG. 53.—Fränkel's pneumonia coccus, bred from the expectoration. Prepared by Prof. Gärtner. Oil immersion lens, one-twelfth; eye-piece No. 4.

But Fränkel's coccus is likewise found in the saliva of many healthy persons (about one-fifth of the healthy), and, according to later examinations, it is not distinguishable from the coccus of sputum septicemia.

It is almost always found in the sputum, and most abundantly during croupous pneumonia, but, as can be understood from what has been said, it likewise appears in catarrhal pneumonia, and also in all other possible sputa, and may be bred from them. From these facts its diagnostic significance is very greatly impaired. It can be confounded with other similar cocci.

Friedländer's coccus is most probably likewise an exciter of croupous pneumonia, but a very rare one. This coccus is, however, not only occasionally found in the sputum of a pneumonic patient, but it or some cocci microscopically similar to it is also found in the most different other sputa.

Of Pio Foà's coccus we as yet know little.

It is to be pointed out here that both croupous pneumonia and forms similar to it may be produced not only by invasion of the cocci of pneumonia, but also by streptococci and pyogenous staphylococci.

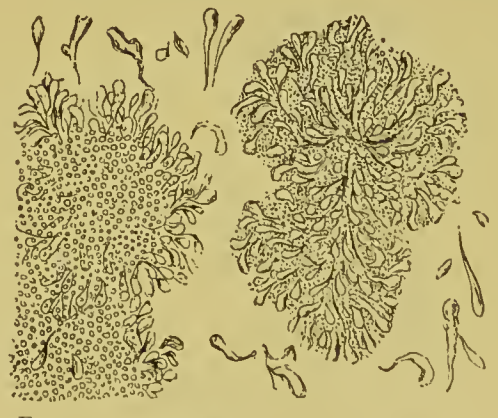


FIG. 54.—Actinomyces (after v. Jaksch).

Method of Staining the Pneumococci.—Fränkel's coccus is stained after Gram's method in dry cover-glass preparations, and best with anilin-gentian-violet solution. From this it is immediately transferred to the decoloration fluid (iodin 1.0; potassium iodid 2.0; aq. destil. 300.0) for two to three minutes, then to absolute alcohol to complete the decoloration. Fränkel's cocci are intensely stained, but occasionally Friedländer's cocci are decolorized. Staining Friedländer's coccus: Dry-cover-glass prepara-

[1] It has lately been found by Pernice and Alessi (*Riforma med.*, 1890) in croupous pneumonia in all possible organs, some of which showed no inflammation at all.

tions are put for a couple of minutes in a 1 per cent. solution of acetic acid. This is then blown away with a pipette; it is dried in the air, placed in anilin-gentian-violet solution for a few seconds, then rinsed in water. Pio Foà recommends Gram's method for his coccus.

Actinomyces.—In actinomycosis of the lungs or of the pleura, in isolated cases, this fungus is found in the sputum. I have observed it in the characteristic small kernels (see page 150). It is recognized by the sort of clubs, closely pressed together, which project from the surface of a confused mass which looks much like detritus. We can best see the club-like projections without staining. The fungus can be distinctly stained by Gram's method.

Of late several persons have succeeded in making pure cultures of actinomyces, but for diagnostic purposes pure cultures will only very exceptionally come into consideration.

Mould (aspergillus, mucor) and isolated yeast-cells, when seen in the sputum, are without significance. The microbe of whooping-cough of Letzerich and Berger still needs confirmation.

Moreover, the sputum always contains a great quantity of bacilli and cocci of all kinds, which no doubt come partly from the mouth and partly, probably, from the upper respiratory passages. The majority of these micro-organisms have no known pathogenic significance. It is of great interest, however, that there are micro-organisms in the sputum which are perfectly identical with certain pathogenic ones, but which in a given case do not produce the morbid phenomena proper to them, and so do not seem to be virulent—as, for instance, Fränkel's and Friedländer's coccus of pneumonia. In this connection is to be mentioned the fact that Löffler has found his diphtheria bacillus persisting in the mucus of the mouth for some time after recovery from the disease.

4. Chemical Examination.—This has a minor place, considered with reference to diagnosis.

There occur in the sputum *albuminous corpuscles* in the form of mucin, nuclein, serum-albumin. The latter is very abundant in edema of the lungs. Kosselt asserts that peptone is found very abundantly in the sputum after the crisis of *pneumonia*. He also states that peptone is found, although in much smaller amount, in every purulent sputum. But this has lately been contradicted by Stadelmann.

Temporary fatty acids occur very abundantly in *gangrene of the lungs* (Hoppe-Seyler, Leyden, and Jaffé).

Finally, it is notable that in *gangrene of the lungs* and *bronchitis* there is found a ferment like the pancreas ferment (Filehne, Stolnikow). According to investigations by Stadelmann, it is present also in phthisical sputum, although with considerably weaker effect. The same author has shown that the question is here probably not concerning an enzym, but about micro-organisms which produce a ferment-like effect.[1]

As a sort of appendix we here add a few words on the use of Koch's lymph in diagnosis. Tuberculin, as is now well known, in certain considerable doses produces in tuberculous patients certain general and local phenomena which are comprehended under the

[1] *Zeitschr. f. klin. Medicin*, Bd. 16.

expression "reaction." This "reaction," and particularly the local signs of it, can be made use of for diagnostic purposes. It consists in certain subjective sensations in the respiratory apparatus (particularly pain), in phenomena pertaining to the sputum, of which we have already spoken on page 162, and in physical signs in the thorax. Although this local reaction is sometimes a very distinct test for the presence of tuberculosis in the lungs, and also for tuberculosis of the most different organs formerly latent, yet we cannot unconditionally recommend its use for purposes of a diagnosis of pulmonary tuberculosis, because it requires doses which in some cases produce an exacerbation of the disease, which sometimes results in accelerating its progress. It is a different matter if tuberculin is applied for a diagnosis of tuberculosis of organs less important to life; for instance, of the bones. Here the procedure seems to have a proper application, but it must be omitted if neighboring organs important to life could be harmed by the local reaction—as, for instance, in tuberculosis of the spinal column, of the skull, and of the pelvis.

CHAPTER V.

EXAMINATION OF THE CIRCULATORY APPARATUS.

EXAMINATION OF THE HEART.

The development of the methods of local examination of the heart is closely connected with the introduction of percussion and auscultation. So we have here also chiefly to thank Laënnec and Skoda, as well as Piorry, Friedreich, Bamber, and Gerhardt.

Anatomy of the Normal Heart.

The heart lies upon the diaphragm, sloping obliquely forward in such a way that its long axis is inclined forward and toward the left. It extends from about 8 or 9 centimeters to the left of the median line (apex of the heart) to about 4 or 5 centimeters to the right of the same (*i. e.* about one and a half finger-breadths to the right of the right border of the sternum—right auricle), so that about two-thirds of the heart is in the left half of the chest and one-third in the right half. Its highest point (the left auricle) is at the lower border of the sternal insertion of the second rib, its lowest point at the upper border of the sixth costal cartilage or the fifth intercostal space (see Fig. 55). The three borders of the heart are formed as follows: the right by the right auricle, the lower by the right ventricle, and the left by the left ventricle. Only a small portion of the latter lies on the anterior surface, much the greater part of which is formed by the right ventricle.

The figure (Fig. 55) shows how the lungs glide over the heart, so that only a small four-cornered portion, belonging exclusively to the right ventricle, is in contact with the wall of the chest. Of the borders of this superficial part of the heart, the one toward the right lies between the middle line and the left sternal line, the upper behind the fourth rib, the left somewhat outside of the left parasternal line. Below, the heart is in relation with the liver in such a way that it overlaps the latter with its lower border. It can be seen from the course of the line *c d*, which indicates the complementary space of the *incisura cardiaca lob. sup. sinistra*, that a considerable portion of the heart which is in contact with the chest-wall would become still smaller if the lung should completely fill the complementary space.

These are the location and extent as they are found in the adult in the dorsal or upright position. With children the heart (as well as the diaphragm and the lower borders of the lungs) is about one rib higher. It is also, since it is proportionately larger, to a larger extent in contact with the wall of the chest; with increasing *age*, on the other hand, it moves lower down, to the lower border of the sixth rib (the

sixth intercostal space) with a smaller portion parietal, since the lungs lie over it to a larger extent. In the side position, especially on the left side, the heart always sinks very considerably to the lower side.[1]

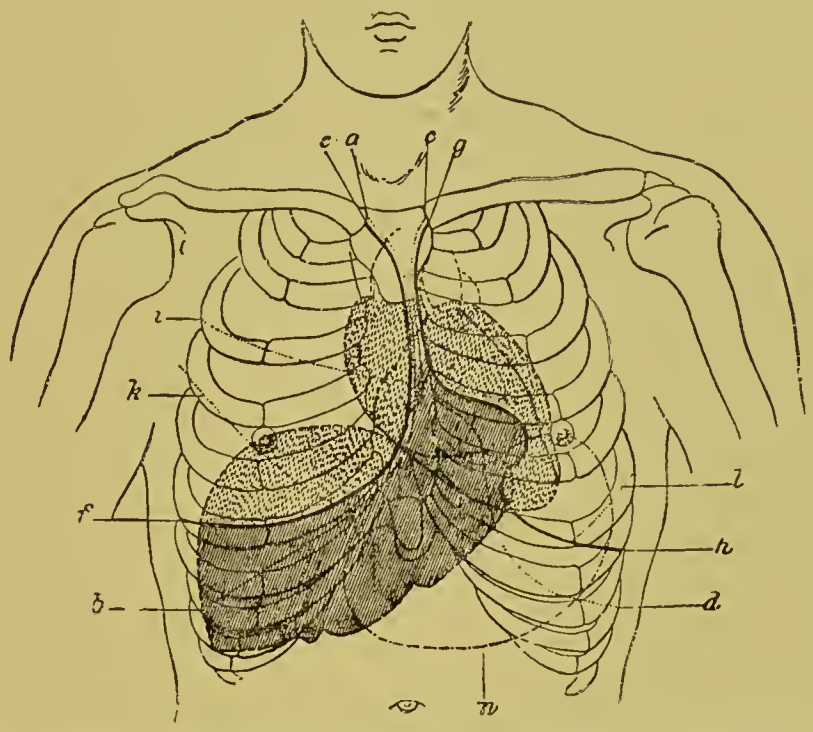


FIG. 55.—Position of the contents of the thorax, of the stomach, and of the liver, from in front (*Weil-Luschka*). The portions of the heart and liver which are drawn with unbroken hatched lines represent the extent to which these organs are in contact with the chest-wall. The portions that are not in contact with the chest-wall, but are covered by the lungs, are represented by broken (clear) hatched lines.

e f, border of the right lung; *g h*, border of the left lung; *a b* and *c d* (. . . .), the boundaries of the complementary pleural sinus; *i*, boundary between the upper and middle lobes of the right lung; *k*, boundary between the middle and lower lobe of the right lung; *l*, boundary between the upper and lower lobe of the left lung; *w*, stomach (greater curvature).

Situs viscerum inversus exhibits the heart in such a way that "right" and "left" are exactly reversed, like the reflection in a mirror. Hence we need not say anything more about it.

PRELIMINARY REMARKS NECESSARY TO UNDERSTAND THE PHYSICAL PHENOMENA OF THE HEART.

What follows is a brief explanation of those facts regarding the physiology and the general pathology of the heart which must be always kept in mind by the educated physician in examining and forming a judgment of the heart:

1. *The Movement of the Blood in the Heart.*—The blood flows from the body through the *cavæ* into the right auricle, whence, during the ventricular diastole, it passes through the right auriculo-ventricular opening, *the tricuspid valve*, into the right ventricle, being urged forward toward the end of the diastole by the weak muscular contraction of the right auricle, and at the same time, in a peculiar manner which cannot be described here in detail, lifts the auriculo-ventricular valves

[1] See under Apex-beat.

so that they are prepared for closing. The systole which immediately follows drives the blood out of the ventricle, through the open pulmonary semilunar valve into the pulmonary artery, the tricuspid valve being at the same time closed. The blood, prevented from flowing back into the ventricle during the diastole which immediately follows by the closure of the pulmonary semilunar valve, passes through the lungs, and from them flows into the left auricle; whence, by the diastole of the ventricle, it flows through the left auriculo-ventricular opening, *the mitral valve*, into the left ventricle, whither it is again assisted at the end of the diastole by the contraction of the auricle. The left ventricle discharges its contents during the systole (the mitral valve being closed) into the commencement of the aorta, through the aortic mouth, which it opens by an actual pressing open of the clack-valve of the semilunar aortic valves; but as soon as the pressure from the ventricular side sinks again, because its diastole begins, then the semilunar valves again close; the blood which has been forced from the ventricle into the *conus aortæ* has its only outlet into the body.

On page 170 are given more details as to the manner in which the contraction of the ventricles takes place, and its relation to the closing of the semilunar valves and the beat of the apex. Our knowledge of these points has recently been greatly extended.

2. *Valvular Insufficiency, and its Effects upon the Movement of the Blood.*—From the foregoing it is evident that the openings of the heart are very important factors, on the one side being the entrance and exit of the ventricles, and on the other being the location of the valves of the heart which hinder any backward flow of the blood. The motion of the blood can only in two ways be interfered with by pathological processes at the openings of the heart—either by narrowing at the opening (*stenosis of valve*) or by the valves losing their power to close (*insufficiency of the particular valve*). Stenosis of a valve may be caused by products of endocarditis, which cause adhesion of the flaps of the valve, with formation of a cicatricial narrowing ring at the base of the valves. Insufficiency may likewise be caused by endocarditis (general shortening of the flaps and of the tendinous processes of the papillary muscles), and this is the most frequent cause of insufficiency; but the condition may also arise from a distention of the opening, so that the flaps are too short to close it (*relative valvular insufficiency* in weak heart with dilatation).

An opening that is narrowed hinders the passage of the blood through it. If it is an auriculo-ventricular opening (*mitral or tricuspid stenosis*), then, at the moment of diastole of the heart, the blood is hindered in its entrance into the ventricles—there is imperfect filling of the ventricles; if it is an arterial opening that is narrowed (*aortic or pulmonary stenosis*), then the exit of the blood from the ventricles is interfered with at the systole. If the valvular mechanism is in such a condition that it cannot perfectly close, then at the moment when it ought to close it allows a part of the blood to flow backward. If the difficulty is with the entrance to the ventricles (*insufficiency of mitral or tricuspid valve*), then with the systole a part of the contents of the ventricle flows back into the auricle; but if the deficiency is at the outlet of the ventricle (*insufficiency of the aortic or pulmonary valve*), then at

the end of the systole, during the diastole which follows, a part of the blood that has just been thrown into the artery will be thrown back into the ventricle.

In one respect all the defects that have been mentioned are alike: they check the blood-current; they cause a stasis of blood in that chamber of the heart which is, with reference to the direction of the blood-current, just behind the defective opening. Thus, a defect of an arterial opening causes stasis in the corresponding ventricle; a defect in an auriculo-ventricular opening occasions stasis in the corresponding auricle, and also beyond this in the corresponding veins.

3. *Compensation; Accommodation of Valvular Deficiency.*—The abnormal resistance which is exerted against the blood-current from the valvular defect would immediately lead to more considerable disturbances of the blood-current if it were not promptly equalized by the increased work of that section of the heart lying (in the course of the blood-current) above the point of resistance. But this does not continue, for with increased work the overloaded section of the heart becomes hypertrophied—*compensatory hypertrophy.* This condition is extremely simple in defects at the aortic opening: they are compensated by hypertrophy of the left ventricle, which is associated with dilatation (*eccentric dilatation*). The latter is especially marked in insufficiency of the aortic valve, and this is explained by the fact that, with aortic insufficiency, during the diastole the left ventricle receives blood from two sources, hence very much more than normal. With mitral insufficiency the auricle must accommodate for the defect; but, notwithstanding the fact that it becomes dilated and hypertrophied, it cannot perform the necessary work, cannot overcome the stagnation: the accumulated blood passes through it to the veins, capillaries, and arteries of the lungs, and so on till it reaches the right ventricle; this becomes dilated and hypertrophied, and thus causes the increase of the propulsive power necessary for the accommodation.

Though defect of the valve of the pulmonary artery is rare, the actual consequences are the same as of defect of the aortic valve; but defect of the tricuspid, which, with the exception of relative insufficiency, is likewise rare, only produces accommodation of hypertrophy of the right auricle, but to a degree hardly to be mentioned, for the increased pressure in the general venous system has no effect upon the pressure in the arteries of the body, and hence cannot produce any notable compensatory hypertrophy of the left ventricle.

Thus, insufficiency and stenosis of the aorta cause hypertrophy of the left, and insufficiency and stenosis of the mitral valve hypertrophy of the right, ventricle. But with mitral insufficiency something more follows: during the diastole of the left ventricle there flows into it from the dilated auricle the blood which has accumulated there under very much increased pressure and in increased quantity; it becomes dilated, and, since it also has to dispose of the increased quantity of blood, which it does by driving part of it forward into the aorta and part backward through the mitral orifice into the auricle, it also becomes hypertrophied. Hence, mitral insufficiency leads to hypertrophy and dilatation of both ventricles.

These different hypertrophies are aids in the diagnosis of the individual valvular lesions.

4. *Hypertrophy of the Heart from Other Causes.*—Besides the valvular defects, certain other conditions lead to hypertrophy: thus, the left ventricle becomes hypertrophied by the increased resistance in the general arterial system produced by *sclerosis of the arteries;* it sometimes results from continued excessive muscular exertion (*idiopathic hypertrophy*), further, from different forms of *chronic nephritis*, and in this it is more marked the longer the general vigor is maintained (hence most marked in renal atrophy); finally, also in *acute nephritis*, if it lasts long enough. The right ventricle becomes hypertrophied whenever there is continued increased resistance in the pulmonary circulation, most regularly and markedly in *emphysema* (from destruction of the capillaries of the lungs from atrophy of the tissue), in marked *contraction of the lungs*, in marked *kyphoscoliosis.*

5. *The form of the heart* is changed in consequence of the hypertrophy (and dilatation): hypertrophy of the left ventricle broadens the heart to the left and somewhat lengthens it; if there is dilatation also, the broadening to the left is still more increased. Hypertrophy and dilatation of the right ventricle simply broaden the heart to the right. Hypertrophy and dilatation of both ventricles broaden the heart in both directions and lengthen it.

6. *Simple Dilatation.*—This results entirely from weakness or paralysis, and is dependent upon a diminished tone of the heart-muscle, with a simultaneous loss of its power to contract. It may also occur in a heart that was previously dilated and hypertrophied, and it then results in a very great enlargement of the heart. In dilatation of the heart the enlargement is nearly symmetrical in all directions.

The diagnosis between enlargement of the heart from hypertrophy (with dilatation) and the dilatation just mentioned is chiefly made by the consideration of the evidences of the amount of work the heart is doing.

7. *The extent to which the heart is in contact with the chest-wall* is in very close relation to the size of the heart.[1] An enlarged heart always has a larger area in contact with the chest-wall than does a normal heart if there are no conditions in the neighborhood of the heart which keep it away from the chest-wall or hinder an increase of its parietal contact. The area of its parietal control may be diminished by emphysema of the lungs or by an increase in the volume of the lungs, whether from anomaly of both lungs or only of the left lung, either chronic or temporary. In every case the distended lungs endeavor, as it were, to put themselves, to some extent or entirely, between the heart and the thoracic wall; that is, to interfere with the parietal position of the heart. In such a case we speak of the *overlying of the heart by the lungs.* In emphysema a smaller area of the heart is in contact with the thoracic wall than when the lung is normal. Likewise, when the heart is enlarged, if at the same time there is emphysema, it does not show itself by a more pronounced parietal position. Hence, when these two conditions exist simultaneously, enlarged heart and emphysema, the

[1] Regarding the pericardium, see later.

heart may be in parietal contact to a degree corresponding to the normal, or even to a more or less diminished area.

Another condition has a contrary influence: this is an inflammatory adhesion of the border of the lung with the parietal pleura at the *incisura cardiaca.* This not only prevents all respiratory movement of the edge of the lungs over the front of the heart, but there is also a recession of the lungs from over the heart through a simultaneous shrivelling of the pleura and of the lungs. Thus, the heart is parietal to a larger extent than is proportionate to its size if the lungs were of normal size. Enlargement of the heart is simulated by the heart having an abnormally large free space in front of it.

Hence, in forming an opinion as to the size of the heart from the extent to which it is in contact with the chest-wall we must always bear in mind the possibility of the presence of these conditions.[1]

Inspection and Palpation of the Region of the Heart.[2]

Both these methods of examining the heart, like the foregoing, will be best practised in a moderately high dorsal position. There are technical difficulties in examining a patient either standing or sitting, but sometimes in severe heart-diseases the latter cannot be avoided on account of the existence of *orthopnœa.* Palpation may be performed either with the tips of the first and second fingers or with the flat, bare hand.

The Apex-beat.—Normal Conditions.—The apex-beat is of the greatest importance as an anatomical starting-point, for it corresponds either exactly to the apex or to a spot very close to it a little nearer to the median line. In the majority of healthy persons it is recognizable by the eye, as well as by the finger applied to the spot, as a rhythmical and systolic projection forward about the breadth of the finger, which in the adult in the upright or dorsal position occurs in the fifth intercostal space just within the mammillary line; only exceptionally, chiefly with persons with very short chest, it is found in the fourth intercostal space. In children, up to the age of ten years, it is usually found in the fourth intercostal space, and either in the mammillary line or just outside of it.[3] In old age, on the contrary, it is sometimes found in the sixth intercostal space. Much fat or the mamma, also narrow intercostal spaces, render it invisible, but yet it may generally be felt. Moreover, without a distinct cause, it may sometimes be entirely wanting in healthy persons.

Quiet breathing produces no change in the apex-beat. With deep inspiration it is covered by the distended lung, which then occupies the complementary space; if it be still evident, it moves sometimes an intercostal space lower down, corresponding to the inspiratory sinking of the diaphragm.

The effect of *change of posture* is very noticeable in the side position: the left-side position moves the apex-beat outward beyond the mammillary line, even as far as the anterior axillary line; the right-

[1] See percussion of the heart: 1. Absolute heart-dulness.

[2] The two methods of examination have such close connection with reference to the heart that to separate them would seem to be artificial.

[3] See above in the section on the Anatomy of the Normal Heart.

side position causes the beat to disappear or moves it somewhat to the right.

Physical exertion and mental excitement, the chief physiological disturbers of the heart's action, may noticeably change the apex-beat in perfectly sound persons, but still more in nervous persons: it may become plainly stronger and even broader or move somewhat to the left.

The physical conditions of the apex-beat and its relation in time to the phases of the revolution of the heart have been for a long time the subject of manifold examinations. Only lately have these questions, as it seems, finally been made clear by Martius. His procedure is as follows: He notes in the curve which he causes the heart-beat to write by a Gunmach's polygraphion the moments of the first and second heart-sounds—*i. e.* of the closure of the auriculo-ventricular and arterial ostia. This is accomplished with almost absolute exactness by simultaneous auscultation, but only under one condition—*i. e.* if the heart beats perfectly regularly. In this case the movements of the marking hands are just as exactly in harmony with the heart-sounds as regards time as in dancing the movements of the feet are in harmony with the notes of the music, or as in playing a duo the movements of the fingers of the piano-player with those of the violin-player. Martius calls his method "acoustic marking method."

The most important thing in the results of these investigations is the irrefutably demonstrated proof of the relation in time of the apex-beat to the systole of the heart. The apex-beat falls about in the middle between the closing of the auriculo-ventricular and arterial valves; and, as Martius farther on, by synchronous observation of the curve of the carotid and that of the apex-beat, for the first time proves exactly that the beginning of the streaming of the blood into the aorta coincides with the highest point of the cardiogram—*i. e.* with the summit of the apex-beat—it follows necessarily that the ascending branch of the curve of the apex-beat, which lies between the closing of the auriculo-ventricular valves—*i. e.* the beginning of the systole and the opening of the ostium aortæ—corresponds with a space of time during which the ventricle contracts, but has not yet reached that external pressure which overcomes the pressure in the aorta, and which thus opens the semilunar valves. Therefore there is a time in this first part of the systole when the ventricle is closed both forward and backward (Verschlusszeit).

The heart-beat is therefore formed during this "Verschlusszeit," which is a refutation of the recoil theory propounded by Alderton, Skoda, and Gutbrod. It is rather the change of form and position of the hardened ventricle which produces the impulse, as Ludwig and others recognized previously, but never exactly proved.

Displacement (Dislocation) of the Apex-beat in Disease.—It may be brought about—(*a*) by dislocation of the heart, (*b*) by enlargement of the heart.

(*a*) **Dislocation of the Heart.**—The apex-beat is a very important sign for determining this, since the other methods often have a very indefinite result or may entirely fail.

Deformity of the thorax may cause displacement in all possible directions. It may happen that in a chest that is flattened or pressed

in in the neighborhood of the heart the apex-beat (likewise the heart) will be found considerably outward or considerably inward.

Emphysema of the lungs, in case the apex-beat is not lost by the overlapping, presses it down into the sixth intercostal space (depression of the diaphragm).

In exudative pleuritis and *pneumothorax* the heart and apex-beat are pushed toward the sound side, in the worst cases as far to the left as the middle axillary line, but to the right very rarely beyond the mammillary line. Likewise, the mediastinum and the base of the heart move over, although not so far as the apex. *Mediastinal tumors* may have the same effect as pleuritis of the right side.

In pleurisy of the right side the apex is sometimes pushed not only to the left, but also upward into the fourth intercostal space. We are not certain why this is so. It is highly improbable that the left lobe of the liver rises up while the right is dragged down, for the point of traction, at the suspensory ligament, brings it still lower by the pressure of the exudation upon the right side. The location of the heart when pressed upon is subject to many disturbances which we cannot describe at this time.

Shrinking of the lungs and of the side of the chest after a pleuritis draws the mediastinum and the heart into the diseased side, and at the same time draws the diaphragm up; hence in shrinking of the right side the heart moves upward and to the right side, but in disease of the left side it is drawn upward or upward and to the left.

If the heart chances to be drawn to the right so much as to bring it under or close up to the sternum, where the intercostal spaces are very narrow, of course we cannot observe the apex-beat.

In *exudative pleuritis* it sometimes happens that the heart becomes fixed by inflammatory adhesions, and then the apex-beat remains in the new position even after the cause of the displacement has been removed.

Elevation of the diaphragm as a result of peritonitis or of simple mechanical pressure from below, or from neurotic paralysis of the diaphragm, causes dislocation of the heart upward or upward and to the left.

(*b*) **Enlargement of the Heart.**—*Hypertrophy* and *dilatation* of the *left ventricle* are made manifest by displacement of the apex-beat *outward* or *outward* and *downward*, and under some circumstances as far as to the posterior axillary line and the eighth intercostal space. The apex-beat is also broader and stronger.[1]

The conditions which bring about hypertrophy and dilatation of the left side have been referred to on page 170. Likewise, hypertrophy and dilatation of the right ventricle displace the apex-beat a little toward the left, since the large right ventricle pushes the left somewhat to one side. But the displacement is always quite small, at most not beyond the mammillary line.

Alteration in the Width and Strength of the Apex-beat.—We judge of the breadth both by inspection and palpation. We seldom have an increase in the *breadth* without an increase in the strength as well: in the normal heart, if it becomes parietal over a

[1] See below.

larger area from shrinking of the lungs; moreover, I have sometimes seen it with deformity of the chest (without hypertrophy of the heart), and where there was marked wasting, so that the patient was very lean.

As a rule, breadth of the apex-beat is associated with a strong beat.

The *strength* of the apex-beat can only be made out by palpation. By constant practice with the hand it can be distinctly recognized. An apex-beat that is so strong that it lifts the finger that is moderately pressing over it is called "heaving."

Temporary, often notably strengthened and moderately broadened, impulse is caused by increased heart-work[1] in consequence of exertion and mental excitement. For this reason, when these two conditions can be excluded, the heart ought always to be examined.

In *nervous palpitation*, *Basedow's disease*, and sometimes in *chronic nicotin-poisoning* the heart-beat may for a time be very much stronger, and even somewhat broader, as an indication of the increased work of the heart, without any organic change in it. The same thing occurs, though in a moderate degree, in *fever*. Moreover, the apex-beat may be stronger if the heart is pressed firmly against the chest-wall, as in mediastinal tumors, although the heart's work is not increased.

Continued strength and *breadth of apex-beat* is the most important sign of hypertrophy of the left ventricle. In well-marked cases the beat is "heaving," and is as wide as several fingers, being displaced toward the left and downward.[2]

It is assumed that an enlarged heart works with strength increased in proportion to its increased volume. If the heart becomes weak, then there is a diminution as regards the breadth and strength, and yet it may be distinctly recognized as diseased.

In many cases it is difficult to separate the apex-beat from the "heart-beat" in general.[3]

Weakening of the Apex-beat.—It has been mentioned already that the apex-beat may be weak in persons who are perfectly healthy, or it may be entirely wanting.

Pathologically, it is diminished or lost—

(*a*) By the activity of the heart being concealed by overlapping; from emphysema of the lungs, by a pleuritic or pericardial exudation, and by tumors.

(*b*) By *edema*, *emphysema of the skin*, inflammatory diseases of the chest-wall in the neighborhood of the heart.

(*c*) By *diminution of the work of the heart*, as takes place with any kind of degeneration of the heart-muscle: here we may mention myocarditis, lipomatosis cordis, weakness or degeneration of an hypertrophied heart, especially with incompensation, with valvular deficiency, weakness in febrile diseases (especially collapse).

The disappearance of an apex-beat which has previously been distinct is sometimes the only sure, and hence is a very important, sign of the development of *exudative pericarditis*. But diminution of the work of the heart is more distinctly declared at the radial pulse than by the apex-beat.[4] Moreover, the radial pulse is the only direct

[1] See above.
[2] See above.
[3] See below, p. 176.
[4] See below for the significance of all these conditions.

measurer of what the heart does in all the above-mentioned cases of concealment of the work of the heart. It is especially important in pericarditis.

Where the apex-beat is covered by fluid in the pericardium it often again becomes distinct when the patient sits up or bends forward, because then, on account of its greater weight, the heart rests against the chest-wall. It is then often found in the sixth intercostal space, because the distended pericardium presses the diaphragm down. This sign, of course, is wanting in cases where the apex-beat is missed from weakness of the heart.

Further, the apex-beat is wanting where there are *pericardial adhesions*,[1] and sometimes in *stenosis of the commencement of the aorta*, and this notwithstanding the existence of hypertrophy of the left ventricle (slow ventricular contraction resulting from difficulty in emptying itself).

So far as experience goes, "*systolic drawing-in*" in the neighborhood of the apex-beat has no diagnostic value.[2]

Doubling of the Apex-beat: Systolia Alternans; Hemisystolia.—There are cases of abnormal activity of the heart where, of two single revolutions of the heart's action, during the first the observer gets the idea that the work is accomplished chiefly, or even exclusively, by the left, and that of the second by the right, ventricle. Unverricht has observed in a mitral insufficiency that in the one contraction of the heart there is a vigorous impulse of the apex, a mitral murmur, and a diastolic aortic sound; in the other, that a weak heart-impulse, more to the right, a diastolic pulmonary sound, and epigastric pulsation were more distinct. Leyden, who first directed attention to this condition, has described cases where the phenomena gave rise to the supposition that there was an alternating contraction of the ventricles; one time both, and the next time only the right one, contracted, so that to one contraction of the left ventricle there were two of the right. The latter Leyden has called *hemisystole;* the former Unverricht denominated *systolia alternans.*

Some believe that in such cases there is simply bigeminate with an alternating relation of the contractions of the heart.[3] We cannot refrain from making the remark here that to-day the terms "bigeminate" and "alternating" action of the heart are frequently misapplied,[4] and that this confusion is increased by Unverricht's denomination, "*systolia alternans*," which, in truth and justly, should correspond with the alternating pulse.

The application of the graphic method to the impulse of the apex (cardiography) has, as has been mentioned before, advanced our understanding of the course of the heart's revolution in a high degree, especially the recent work of Martius. But as regards pathology, and especially for diagnostic purposes, this method has not yet produced any results worth mentioning.

The Neighborhood of the Heart in General.—*Prominence of the neighborhood of the heart*, bulging, including the ribs and sternum,

[1] See below under Systolic Retraction.
[2] Regarding systolic retraction of the entire region below the heart, see below.
[3] Compare under Examination of the Pulse.
[4] Compare under Pulsus Alternans and Bigeminus.

takes place gradually in marked hypertrophy and dilatation: in hypertrophy of the right ventricle or of the right and left the bulging sometimes extends to the right side of the sternum; in hypertrophy of the left ventricle alone it lies more to the left. *Pericarditis exudativa* sometimes causes a distinct swelling.

This sign depends upon two factors—the size of the heart or of the pericardium, and the flexibility of the chest-wall. If the latter is marked, the swelling develops quickly, as in acute pericarditis, and is very marked (enlargement of the heart in children); when the thorax is rigid there may be no projection, though the heart is very large. This condition is not to be confounded with the pressing forward of the heart from mediastinal tumors or aneurysm.

Generally when there is a *broad heart-beat* in the intercostal spaces in the neighborhood of the heart, and even upon the ribs and sternum, it is from a hypertrophy of the heart. But also, when there is contraction of the left lung, with the heart free from attachment, the motions of the heart may be seen as well as felt over a broader extent in the intercostal spaces. If in such cases the heart's action is excited, there is the impression of a notable hypertrophy of the heart, even when the heart is quite normal in size.

If the heart, from dilatation or retraction of the lungs, is more extensively parietal, weakness of the heart occurs, then we not infrequently see a broader waving in the intercostal spaces, and even on the ribs of the precordial region, which, however, by its evident lack of energy, is visibly in contrast with its former circumscribed but powerful motions.

Martius has sometimes seen, in simple over-exertion of the heart by bodily activity, not only the above condition, but even an extremely strengthened apex-beat, and visible and palpable heaving of the whole precordial region, with synchronous weakness of the pulse. He considers this contrast between visible labor of the heart and the pulse to be pathognomonic of pure over-exertion of the heart, provided stenosis of the aorta or aneurysm can be excluded.

Pulsations at the base of the heart sharply limited to the second intercostal space on the right and left sides of the sternum come from the aorta or pulmonary artery. They are rarely visible; generally they can only be felt. If they are systolic, they may indicate aneurysm of these vessels. More frequently we may feel a diastolic shock, especially upon the left, over the pulmonary artery. If the lungs and heart are normal, it cannot be felt; but if the lungs are drawn back from the base of the heart (by shrinking or by enlargement of the heart), or if there is thickening, then it may be felt, especially if it is simultaneously strengthened by hypertrophy of the right ventricle. In emphysema of the lungs there exists the peculiar condition that, although the closure of the pulmonary valve is in a marked degree stronger, yet it cannot be made out because the inflated lung lies over it.

Pulsation in the region about the heart occurs in empyema located near the heart upon the left side (empyema pulsans); farther, in *aortic aneurysm*.[1]

[1] See this.

Although *systolic drawing-in at the apex of the heart* is of no significance,[1] yet systolic drawing-in of several intercostal spaces in the neighborhood of the heart, but especially of the ribs and the lower part of the sternum, is of diagnostic value: it is probable that there is *pericarditis adhæsiva* with *mediastinal pericarditis*, accompanied by thickening. But yet these signs may be entirely wanting, although the condition is present; and, on the other hand, they may be observed in cases where this condition does not exist. The drawing-in may be caused by a dense mediastinum being adherent to the spine, and again by pericardial adhesion to the chest-wall; its contraction—that is, its constantly becoming shorter—must of necessity cause a drawing-in of the chest-wall.

"*Buzzing*" and friction-sounds that can be felt in the neighborhood of the heart accompany very marked endocardial or pericardial sounds.[2]

The Epigastrium.—In inspecting and palpating the heart this must always be considered. *Systolic trembling*, or even *systolic pulsation*, may be observed here if the heart, more particularly the right ventricle, is drawn nearer the abdominal wall by the depression of the diaphragm, but especially is this the case when, *at the same time*, the right ventricle is hypertrophied: in emphysema of the lungs.

This epigastric pulsation must not be confounded with that which is to be seen from the abdominal aorta when the abdomen is very empty and the abdominal wall very thin, whether the aorta pulsates normally strongly or not, or whether or no there is an aneurysm of the abdominal aorta. This pulsation is, moreover, best transmitted when a tumor of the lymphatic glands, of the stomach, or a thin but firm liver lies over the aorta. Sometimes (not always) the pulse is felt noticeably later than the systole of the heart.

Percussion of the Heart.

This has for its object the determination—

1. Of the absolute "small" *dulness of the heart*, which corresponds with the portion of the heart that is in contact with the chest-wall, and which has an almost definite relation to the size of the heart.

2. The so-called *relative heart-dulness*, which lies above and to the left of the absolute dulness, and which is determined by the thinness of the lungs around its border.[3] It often stands indirectly in some relation to the size of the heart, but only exceptionally represents the real size of the heart.

To these two Ebstein has recently added—

3. *Palpatory percussion of the "heart's resistance,"* which is determined by ascertaining the anatomical size of the heart.

Normal Percussion Figure of the Heart.—Methods of Percussion.—1. **Absolute Heart-dulness.**—This is determined by light percussion, and corresponds, in fact, to the portion of the heart that is parietal. In two respects it departs from this, though not essentially: the small strip of the heart which is parietal behind the sternum

[1] See above.

[2] See under Auscultation of Heart.

[3] See above, p. 108.

between its left border and the inner border of the right lung is not dull, as would be expected, but gives a clear sound, as indeed occurs over the whole surface of the sternum;[1] the lingula, being so small, does not affect percussion; over it we notice absolutely deadened sound. Thus we have the following figure of the absolute heart-dulness in persons in middle life (Fig. 56): the boundary on the right is the left sternal line; the upper boundary lies upon the fourth rib; the left boundary is outside of the left parasternal line. The lower boundary toward the liver cannot be exactly determined, it being defined by the apex-beat, and generally also by the upper border of the sixth rib. In children the area of heart-dulness (absolute) is somewhat greater, the heart being relatively larger: the upper boundary in the third intercostal space; hence the apex-beat is generally in the fourth intercostal space; the left boundary near the mammillary line; in old age, however, it is smaller (from inflation of the lungs), about over the fifth rib or the parasternal line.

In quiet breathing the dulness does not distinctly change; in *deep inspiration* it is very decidedly diminished or entirely disappears,

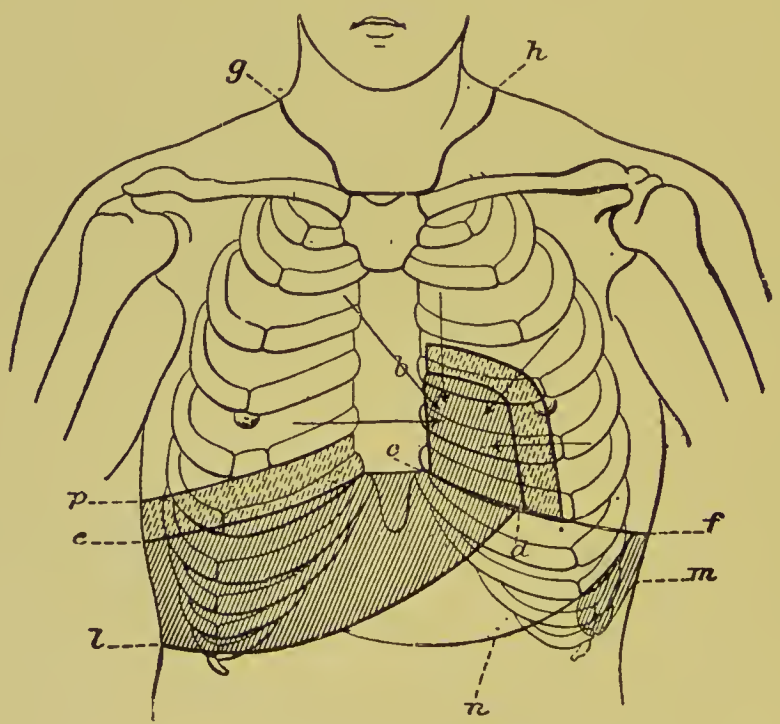


FIG. 56.—Percussion-boundary of the lungs in front (Weil):

g h, the upper limits of the lungs; *e f*, the lower limits of the lungs; *b d*, boundary between the lungs and heart at the incisura cardiaca. The strongly-hatched surface represents the portions of the heart and liver that are in contact with the wall of the chest; the lighter hatching, the so-called relative heart and liver-deadness (see later); *m*, spleen-deadness.

because the costal cartilages come close together at the sternum.[2] It makes no difference whether the examination is made in the dorsal or the upright position. Examination upon the side makes considerable alteration of the area of dulness.

[1] See above, p. 106.

[2] Compare the course of the boundary of the complementary space (Fig. 55).

The beginner is apt to be much confused, because sometimes in a considerable part of the location of heart-dulness, even within the entire region, he will find a tympanitic-deadened resonance which downward, but without a distinct limit, changes into a pure tympanitic resonance. This is especially frequent in short persons with a short, thick thorax and a full abdomen. The resonance is from the stomach, which lies under the heart, and is more promptly elicited by strong than by weak percussion. When there is an otherwise normal condition of the heart and lungs this phenomenon has no pathological significance.

It is very difficult to judge of the absolute dulness of the heart, particularly in pathological cases: it denotes the parietal position of the heart, which, however, depends not only on the size of the heart, but also on the size of the lungs, the latter of course in a reversed sense. This circumstance makes it very difficult to form a conclusion as to the size of the heart from the extent of the absolute dulness; however, any one who, in judging of the percussion figure of the heart, accustoms himself every time to take exactly into consideration the condition of the lungs minimizes to a great extent every difficulty. But in many cases the result of the examination still remains entirely in doubt.

2. **Relative Heart-dulness.**—This forms a border around the absolute dulness to the left and above it, and it corresponds with the thinned-out portion of the lungs. It is revealed by stronger and, in its upper part, by comparative percussion. It no doubt depends, within certain limits, upon the delicacy of the perceptions of the individual making the examination as to where he will fix the limits between it and those of normal lung-sound; for, in the first place, on account of the very gradual transition of the sound, the individual perception of hearing is a large factor here; in the second place, according to explanations already made, the size of this relative dulness depends upon the degree of intensity of percussion, for which degree we unhappily do not have an absolute measure. One who percusses very carefully and, above all, always with about equal force, will accustom himself by and by to a somewhat equal judgment of the relative dulness of the heart in one case as compared with another. And, again, for determining the size of the heart the relative dulness has an important, and not seldom a greater, value than the absolute dulness. But it is extremely rare that two different examiners agree about the exact limit of the relative dulness of the heart, and for this reason there are great difficulties in giving instruction on this point.

It demands, therefore, much practice, uniformity of method of instruction, agreement in the perception of sound and of the resistance, rightly to estimate the relative dulness of the heart. But on account of the imperfectness with which the absolute dulness of the heart is determined it is important to give attention and pains to the relative.

How different individual examiners are in regard to this point may be learned from the fact that some—as, for example, Reiss—think that it is possible to produce by strong percussion the projection of the anatomical figure of the heart upon the thorax. In our opinion, how-

ever, that is much too strong a statement, except with regard to individual cases of very delicate thorax.

According to Weil, the line of relative heart-dulness is as follows (see Fig. 56): It begins above at the lower border of the third rib, continues in a curve downward toward the left, within the mammillary line. In rare cases, especially in fat persons and those with a short sternum, there is also a relative dulness at the right of the absolute dulness, which is limited by the lower end of the sternum. *In children* the relative dulness begins at the upper border of the third rib, even in the second intercostal space; it extends somewhat beyond the left mammillary line, and is also constantly present on the right, and, indeed, reaches even beyond the right side of the sternum.

Opinion is divided regarding Ebstein's newer method of determining by direct palpatory percussion *the resistance of the heart* as the true image of the exact size of the heart. Indeed, Eichhorst is the only one who warmly espouses the idea. It seems to me that there is no doubt of its use in many cases—that is to say, in those with delicate thorax having thin covering of flesh. At the same time, I cannot recommend it as a subject for instruction to others, since it is liable to give rise to many mistakes, and in my opinion it is very difficult to learn.

3. **Method of Percussing the Heart.**—We percuss strongly on both sides close to the sternum, going downward, and note the upper boundary of relative heart-dulness; then we percuss lightly the upper boundary of absolute heart-dulness; next we percuss upon the outer ends of radii drawn from the middle of what is thought to be an area of absolute dulness (first the one obliquely upward to the right, then from the right, always beginning beyond the sternum; then on the left obliquely upward; lastly, from the left), always strongly at first to determine a possible relative dulness, then lightly for the absolute. At first we percuss at longer intervening spaces, of at least 1½ centimeters, and, when a difference of resonance is found, then at short intervals of space over the particular region. In Fig. 56 the lines and the directions in which we ought to percuss are designated by arrows.

For ascertaining the size of the heart in difficult cases, particularly if the heart is overlaid by emphysematous lung, Gumprecht has lately recommended to percuss while the patient bends forward. He found that in this position the area of dulness became larger, more intense, and more resistant. The boundaries ascertained in this way, in general, were as follows: the fourth rib, the left edge of the sternum, the left mammillary line; in enlargement of the heart the dulness extended over a larger area with the patient in the forward position, even if it was diminished when lying upon his back.

Enlargement of the Area of Heart-dulness. — Generally, relative dulness and absolute dulness exist in about equal proportions, but now and then the relative may be very small. Always in enlargement of the right side of the heart, and sometimes in enlargement of the left side, relative dulness toward the right is increased as compared with the absolute.

Heart-dulness is increased—

1. *In hypertrophy and dilatation of the heart.* If of the right ventricle, the dulness spreads toward the right, sometimes also slightly

toward the left, the whole involving a half-circle. If the left ventricle is changed, the increased dulness is toward the left and downward, not infrequently also upward, but scarcely any, or at most very little, toward the right.[1]

2. *In dilatation of the heart* (weak heart). This causes the previously existing dulness, it may be of a normal heart or of one that was already hypertrophied, to spread out on both sides. (For distinguishing from hypertrophy, see "Apex-beat" and "Radial Pulse.")

3. *Fluid in the pericardium* (*pericarditis exudativa and hydropericardium*). Generally, this causes the dulness to enlarge, at first upward and then to the right and left. Not infrequently the area of dulness has a three-cornered shape—one point above close to, and on the left of, the sternum, one below on the other side of the sternum, and one on the left, also below, on the outer side of the mammillary line; the relative dulness is generally very small. If the exudation is very large, the lung surrounding it is generally retracted, and hence around the dulness there is a border of tympanitic resonance. In sitting up the area of dulness is greater than when lying down, and when bending forward still greater than in the sitting posture, because there is a change in the extent of area which is parietal.

Regarding the apex-beat in pericarditis, see page 174: in the latter disease it is often deeper and not on the left border of the dulness, as in enlarged heart, but farther toward the right, and generally within the mammillary line (a not unimportant point in differential diagnosis). The pulse[2] is often important.

4. *When the heart is normal, but is to a greater extent parietal on account of retraction of the lung.* In this case the mobility of the border of the lungs in deep breathing is completely wanting. The apex-beat may be normal, but by simultaneous displacement it is farther to the left.

5. *Apparent enlargement of the heart* is noticed if anywhere in its neighborhood there is a diseased condition which causes absolute dulness. Of this kind we may name thickening of the lungs, of the pleura, of the mediastinum, and especially aneurysm. It is almost impossible to mark the boundary between the heart and such pathological structures, since we are denied the aid of percussion; on the other hand, an approximate determination may often be attained during auscultation by the appearances of motion (apex-beat, etc.), and sometimes by the vocal fremitus.

Pulsating affections give especial difficulty, as aneurysm and the empyema pulsans previously mentioned. Here the object is sometimes attained by repeated examinations. For distinguishing empyema pulsans from aneurysm, see the latter.

Diminution or Loss of Heart-dulness.—This takes place—

1. In *emphysema of the lungs*. It affects the parietal conditions of the heart, whether it is normal or enlarged. If the heart is normal, there is considerable diminution of the area of dulness, even, possibly, to its entire disappearance. If the heart is, at the same time, enlarged (as, as has already been mentioned, it generally is in consequence of the

[1] Regarding a small independent dulness which is sometimes found on the right near the upper end of the sternum, see Aorta, p. 219.

[2] See Pulse.

emphysema, which causes hypertrophy of the right ventricle), the emphysema makes the dulness smaller than it would be with a heart of the same size and normal lungs. Hence, when there is emphysema we must make some addition to the extent of the dulness we are able to map out before we form a judgment regarding the heart. A normal area of heart-dulness, with the existence of a marked emphysema, indicates considerable hypertrophy of the heart if there is no adhesion of the borders of the lungs; hence we must notice their active mobility.

2. In *pneumopericardium* entrance of air into the pericardium, either from without by an external injury or from within by perforation of the esophagus, stomach, or intestine, we may have the condition of pneumothorax. There is then tympanitic or abnormally loud and deep resonance in the neighborhood of the heart (also metallic heart-sound); finally, very rarely in *emphysema of the mediastinum*.[1]

Displacement (Dislocation) of the Heart-dulness.—This, of course, arises from displacement of the heart, as has been mentioned when speaking of the apex-beat; but in this case, for various reasons, percussion is generally an imperfect means of determining such change. For one thing, it often happens that the condition which causes the dislocation itself presents dulness, which invades the region of heart-dulness.[2] This is the case when shrinking of the pleura or lungs distorts the heart. Again, it is usually especially difficult to determine the location of the heart by percussion if there exists a vicarious emphysema on the left side simultaneously with considerable shrinking on the right. In this case the heart is sometimes moved over to the middle of the thorax (*mesocardia*).

Still further, the extent to which the heart is parietal is frequently changed by dislocation: thus, when the diaphragm stands very high the heart is pushed upward, usually causing an increased area of dulness, since the heart is then more flat against the chest than is normal.

If there is an *apex-beat* in such cases, it is a very sure sign; but often it is necessary to employ auscultation to aid in establishing by the location of the greatest intensity of sound, at least approximatively, the position of the heart.

Auscultation of the Heart.

Methods and Normal Condition.—Methods.—Ordinarily we are to auscultate the heart exclusively by the stethoscope. After long practice and experience the examiner may think it advisable to compare what he hears with the stethoscope in individual cases—as, for instance, in pericarditis—with the results of direct auscultation; but these are exceptions. The very urgent reason for the use of the stethoscope is that by it we are able to distinguish as sharply as it is possible to do the impressions of sound which come from the different points, so as to be able to refer every sign to its proper place of origin.

First of all, we are to examine the patient when he is in the greatest possible quietude of body and mind: in some cases we may then, after we have begun, find it advantageous to increase the activity of the

[1] See p. 50.

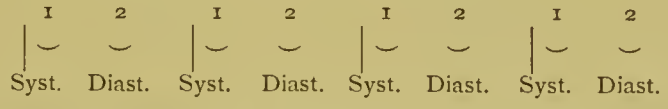
[2] See above, p. 181, under 5.

heart by having the patient make a certain amount of exertion (as by sitting up in bed several times in succession or moving about), since we can thus sometimes obtain certain signs clearer. This will be referred to from time to time. The position of the patient during the examination will, in general, be the same as for percussion, already referred to. However, we often hear much plainer in the upright position, and hence in doubtful cases auscultation in this position is not to be neglected.

More than anywhere else, in auscultation of the heart it is necessary to examine several times. The rapidity and strength of the heart's action and possible extraneous sounds have a great influence upon the distinctness of what is heard. In severe diseases of the heart, especially with heart-failure from different causes which will be mentioned, the impression is generally so confused that no physician of experience will pronounce a definite opinion until, by appropriate treatment, the heart has been restored to a degree of strength.

Normal Condition.—Over the whole region of the heart and for a certain distance beyond it we hear, corresponding with each pulsation of the heart, two "tones:" one coincides with the ventricular contraction, the "*systolic*," the "*first*" tone; and one, which is heard not exactly, but still approximately, at the beginning of the diastole, the "*diastolic*," the "*second*," tone. Corresponding with the greater duration of the diastole, the pause between the second and the following first tone is always greater than that between the first and second.

The *rhythm* in general is as represented here:



The apex-beat coincides, to a certain extent, in time with the systolic sound, and likewise, as we can directly observe, with the pulse in the common carotid in the neck. But the pulse of the peripheral arteries occurs noticeably later, so that the radial pulse is felt between the first and second sounds of the heart.

The expression "tones" is not to be taken in a strictly acoustic sense. In reality, it is a short, sharply-defined noise which only approaches a tone. But the term is not so inappropriately selected, as every one must be impressed who compares these phenomena of sounds with the peculiar heart-sounds to be spoken of hereafter.

Both of these *heart-sounds*, the first and second, can be heard over the whole region of the heart, but at different points they are of different nature and origin, as is partly declared by the character of their tone. According to the view now almost universally held, a part of each sound has its origin in each of the four portions of the heart, and hence is in all eight-fold:[1]

1. The sudden tension and closure of the mitral and tricuspid

[1] A short time ago Geigel returned to the opinion formerly held, according to which there are only four heart-sounds, and that on account of new considerations this idea is worthy of notice. As we have only very recently seen Geigel's explanation, we must suspend judgment on this question, and hence, for the present, adhere to the opinion already expressed.

valves causes a systolic sound, which naturally is most distinctly heard in the neighborhood of these valves or over the ventricles.

2. The closure of the semilunar aortic and pulmonary valves causes a diastolic flapping tone, heard most distinctly over those valves or in their neighborhood.

3. The sudden contraction of the ventricle causes a dull systolic sound of short duration.

4. The sudden filling of the *conus arteriosus*, aortic and pulmonary, in consequence of the motion of the blood, or, more probably, of the sudden tension of the walls of these vessels, causes a short, somewhat ringing sound.

Although after the careful investigations of Krehl one might be inclined to consider the first ventricle-sound as only a muscle-sound, it seems to us that it is impossible to entirely exclude the auriculo-ventricular valves and their tendinous fibers from a participation in the production of the first sound. It is difficult to comprehend how the undoubtedly sudden closure of these valves and the just as sudden stretching of their tendon-fibers should take place without producing some sound; but also the weakening of the first sound, even to entire disappearance, which happens in insufficiency of the mitral valve, indicates that the closing of the auriculo-ventricular valves participates in causing the first sound.

Thus, we see that the valves have a very essential part in the production of the heart-sound; and since, as has already been remarked in the "Preliminary Observations" [page 167], the heart-sounds arising in certain circumstances are *only* connected with the valves or the different openings, these are the chief consideration in auscultation. Hence we have chiefly to attend to the auscultation of the *mitral valve*, the *mitral orifice*, the *aortic valve*, the *aortic orifice*, etc.

Hence it follows that we always first listen at those four points of the chest which lie nearest to these valves. But experience has shown that for two of them this is not the best method, as is easily understood from the anatomical relations.

We cannot auscultate the aortic valves at the point of the chest which lies nearest to them, since they are obliquely behind the pulmonary valves, and at that point the sound which comes from the pulmonary artery and its valves predominates; hence we must auscultate at the beginning of the aorta; and likewise we do not ordinarily hear the mitral sounds most distinctly at the point where the valve is located, since a layer of lung there covers the heart, and also because the right ventricle lies in front of the left, but we hear it better at the apex of the heart. The points of election for auscultating the heart are as follows (compare Fig. 57):

Mitral valve,
Left auriculo-ventricular opening, } apex of the heart.

Tricuspid valve,
Right auriculo-ventricular opening, } over the sternum.

Aortic semilunar (ost. aort.): second intercostal space, right of sternum, and for aortic insufficiency the middle of the sternum itself, and likewise the fourth intercostal space on the left side close to the sternum.

Pulm. semilunar (ost. pulm.): second intercostal space, left of sternum.

The accompanying figure exhibits the situation of the openings and the points where they may be best auscultated. We see that the auscultation-points of the mitral and aortic valves are so related to the respective openings that they lie downward from them with reference to the normal course of the blood-current.

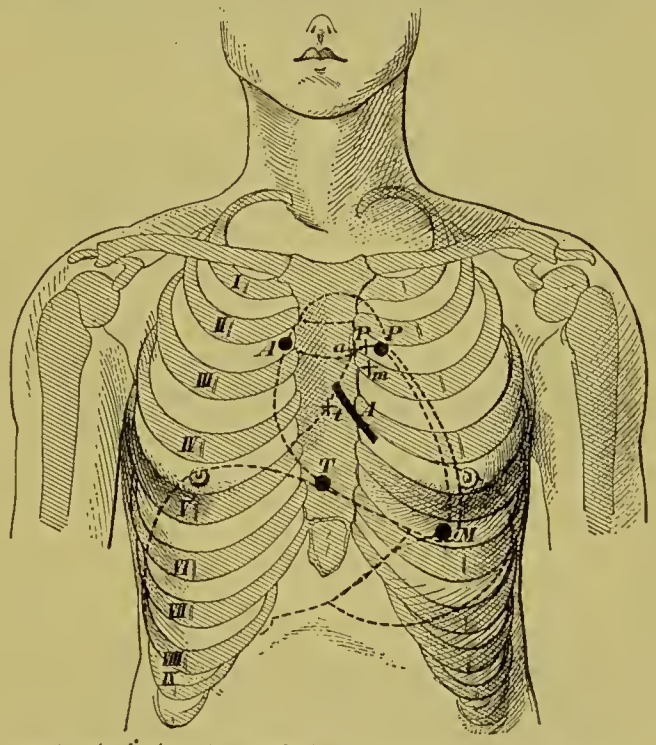


FIG. 57.—The anatomical situation and the points for auscultating the valves of the heart and its orifices. The crosses indicate the anatomical situation, but the black points and lines the places to auscultate. The small letters show the location of the valves, the large ones the points for auscultating: aA = the aorta; mM = mitral valve; pP = the pulmonary orifice; tT = tricuspid.

The "sounds" that can be heard in health at the four points mentioned correspond with the occurrence of the sounds just referred to in the following way:

Apex of the heart (*mitral orifice*):

1st sound: Closure of the mitral valves and ventricular contraction.

2d sound: Prolonged aortic second sound (closure of aortic valve).

Under the sternum (*tricuspid orifice*):

1st sound: Closure of the tricuspid valves and ventricular contraction.

2d sound: Prolonged pulmonary second sound.

Second intercostal space, right or left (*aorta, pulmonary art.*):

1st sound: sudden filling of the beginning of the aorta, of the pulmonary artery, and continuation of the first ventricular sound.

2d sound: closure of the semilunar valves of the aorta or of the pulmonary artery.

Thus, the first sound is a mixed one, composed of muscle, valve, or also of vessel-sound; it is dull and somewhat prolonged. The second sound is throughout wholly from the semilunar valves; it is short, flapping. Hence I represent the first by a dash, the second by a short curved line. The heart's action is hence represented in the following way:



[1] S = systole. [2] D = diastole.

FIG. 58*a*.—Representation of normal heart-sounds.

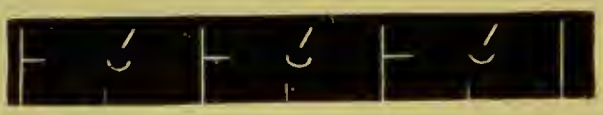
and since we hear the second sound over the ventricle only as conducted from above against the current of blood, over the ventricle it is very light; *hence the accent at the apex* and [*over the sternum, i. e.*] *under the sternum* is represented as follows:



FIG. 58*b*.

In auscultating, however, at the mouth of the arteries we hear the second sound at the place of its origin; it is here louder, and indeed

either



[3]

or

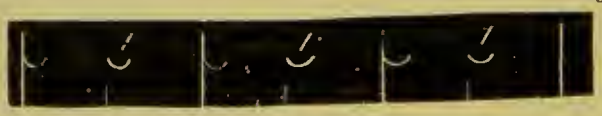


FIG. 59.

[3] This representation departs from the habit of authors, who draw the comparison with the trochaic and iambic foot, and this does violence to the length of the sounds merely for the sake of making the comparison. I maintain that the above representation is more in accordance with the facts.

louder than the first, and hence the accent is *at the base of the heart:* according as the first sound is like the ventricular sound or not.

Differences or Variations within Normal Limits.—*The absolute strength* of the heart-sounds varies very much in persons in health. It depends upon the elasticity and delicacy of the thorax: children and persons with delicate thorax generally have loud heart-sounds; with the former, they are widely conducted by the lungs, and this for the same reason that with them the breathing-sound is sharper.[1] Further, the thickness of the covering of the chest has its effect: large mammæ, thick layer of fat, weaken the sounds. Temporary

[1] See this.

excitement of the heart may increase the sounds so very much that even an experienced person may be tempted to suppose that they are increased by pathological conditions.

The *tone* of the heart-sounds also varies: with many the first sound as well as the second is more "tone-like," with others less so. Especially variable are the first sounds—sometimes shorter, sometimes longer, noise-like, "impure;" further, sometimes very deep and not clear, "dull."

The first sound of the heart (much more rarely the second) may even in health be doubled:

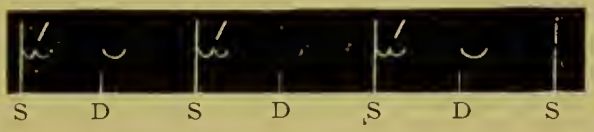


FIG. 60.—Normal first sound doubled.

This is generally only at the end of expiration and the beginning of inspiration, probably disturbed by the ventricles not contracting synchronously (see also under Pathological Doubling).

Regarding the measurement of the intensity of the heart-sounds, see below.

Pathological Changes in the Heart-sounds.[1]— *General strengthening of the sounds* causes one to infer that there is increased activity of the heart: this may occur, as above indicated regarding healthy persons,[2] but to a still higher degree from *temporary excitement* in nervous disease of the heart, and also in Basedow's disease; it is also a frequent accompaniment of fever; this strengthening then indicates increased work of the heart-muscle, without the heart-muscle being necessarily hypertrophied.

Furthermore, in a normal anatomical condition of the heart-muscle the sounds may be strengthened if a healthy heart lies free in an abnormally large space, as in shrinking of the lung. But it must be observed that here there is occasionally hypertrophy of the right ventricle. Also consolidation of the lungs in the neighborhood of the heart causes the heart-sounds to be abnormally loud. Finally, a strengthened and flapping character of the heart-sounds is not infrequent in *anemia*, and particularly in *chlorosis*.

To these cases of simple strengthening of the heart-sounds others stand in opposition, where the phenomenon is combined with the signs of hypertrophy of one or both ventricles, and therefore the question of hypertrophy of a ventricle must be carefully considered in each case of strengthened heart-sounds. Strengthened heart-sounds are, as a matter of course, heard over a larger area beyond the heart than normal. They may be heard over the whole thorax. However, such more *extended perception* of heart-sounds may be due to condensation of the lungs (pneumonia, chronic contracting phthisis).

It is difficult to measure exactly the strength of the sounds of the heart. Recently a very ingenious method has been proposed by H.

[1] It is recommended to arrange in a schedule the pathological conditions of the heart revealed by auscultation, and likewise those discovered by percussion.

[2] See preceding page.

Vierordt. Its significance will be greatly affected by the changing deadening effect of the chest-wall and its covering, also of the lungs. It is interesting to note that normally the mitral first sound is the loudest and the aortic first sound the softest. Dull sounds which, by the usual mode of auscultation, the ear is accustomed to consider light, by this method sometimes manifest themselves as louder, like flapping, although to the ear the latter sound more intense.

Strengthening of Separate Sounds.—Strengthening of a second sound (more emphatic closure of the semilunar valves), if persistent, is a very sure sign of hypertrophy of the corresponding ventricle.[1] Only we must not consider a slight emphasis of the aortic or pulmonary second sound as a pathological strengthening.[2] Abnormally strong, *accentuated pulmonary second sound* is thus a very important *sign of hypertrophy of the right ventricle*, and it is the more important since in this condition percussion is often doubtful. *Strengthened aortic second sound*, especially in sclerosis of the aorta, becomes slightly sonorous, ringing. In hypertrophy of the left ventricle from insufficiency of the aortic valves accentuation is wanting, because in the main the second sound is wanting, since the valves do not close.

This accentuation of the second sound immediately disappears when the heart becomes weak, when heart-failure takes place. The disappearance of the accentuation of the pulmonary second sound is therefore of especial diagnostic value, since we have no other direct sign of commencing failure of the right ventricle. If there occurs a *relative tricuspid insufficiency* from a high degree of weakness and dilatation of the right ventricle,[3] then the pulmonary second sound almost entirely fails, since the blood now has an outlet upon both sides—backward through the ostium venosum and forward into the pulmonary artery, and thus the pulmonary pressure falls off very greatly.

In making his observations in a case of disease of the heart the importance of the second pulmonary sound cannot be too strongly impressed upon the beginner: it is a measure of the activity of the right ventricle, as the pulse is of the work of the left.

Not infrequently *both pulmonary sounds* (much less frequently *both aortic sounds*) are *strengthened* by the base of the heart being in contact with the chest-wall when there is shrinking of the lungs. An accented pulmonary second sound from hypertrophy of the right ventricle may be thus *felt* as a diastolic stroke in the left second intercostal space. Also in mitral insufficiency with hypertrophy of the left ventricle and shrinking of the lung an aortic second sound may be *felt* in the right second intercostal space.

Pathological strengthening and flapping character of the *first sound at the apex* are so frequently occurrences in *mitral stenosis* that to the experienced observer they have diagnostic value. The phenomenon is ordinarily explained as being a consequence of diminished filling of the left ventricle which follows from the lessened size of the orifice by which it is filled; the segments of the mitral valve at the end of the diastole are still very lax, and so come together with more energy at

[1] For the conditions which lead to hypertrophy of a ventricle, see Preliminary Remarks, p. 167.

[2] See p. 186.

[3] See Preliminary Remarks, p. 167.

the beginning of the systole. This explanation does not appear to us to be wholly acceptable.

Weakness of all the sounds of the heart (more inclined to concern the second sound) occurs in *all cases of weak heart*, as takes place in hearts previously sound in consequence of over-exertion, severe hemorrhages, carbonic-acid poisoning, or any kind of interference with breathing,[1] any other kind of poison, as heart-poison in acute febrile diseases; finally, in central or peripheral paralysis of the vagus, as follows disease of the heart-muscle, or as generally at last from some cause or other overtakes an hypertrophied heart.

Hypertrophy of one division of the heart is, as referred to in the Preliminary Remarks,[2] generally "*compensatory*"—that is, it is said to accompany any obstruction of the circulation. If a hypertrophic heart can no longer meet the demands made upon it, we then use the term "*incompensation*." Then heart-sounds that in part were previously strengthened at first become about normal, and then become weaker than normal.

Moreover, when *an emphysematous lung forms a layer over the heart*, the heart-sounds are found to be persistently weakened, even to marked indistinctness, and this involves also the pulmonary second sound, which in emphysema is strengthened. This weakening occurs with large *pericardial exudations* or *hydropericardium;* more rarely from a *tumor* or *pleural exudation* pressing against the heart.

Weakening of Individual Sounds.—If there is an "organic heart-murmur,[3] then the sound with which it occurs or at which it ceases becomes either weakened or indistinct, or it is entirely wanting, so that the "murmur" takes the place of the sound. But also with certain valvular defects there occurs weakening of other sounds: of the *aortic second sound in mitral stenosis*, in consequence of which the left ventricle has only a little blood to throw into the aorta;[4] weakening of the same *aortic second sound in stenosis of the aorta;* also the *pulmonary second sound in stenosis of the pulmonary artery*, as a consequence of those valves being less free in their action. Not without diagnostic value, also, is a high degree of weakening *of the first sound at the apex* in *aortic insufficiency*. This is explained by the reflux from the aorta, with the normal afflux from the auricle, filling the ventricle abnormally full: it becomes dilated, and thus the tips of the mitral valves, even before the beginning of the systole, are somewhat pushed up. When the systole takes place there is then only a moderate increase in its tension. Moreover, in aortic insufficiency, over the aorta the first sound is often weak and very impure, without other contemporaneous signs of aortic stenosis being present.[5]

Alteration in the rhythm of the heart-sounds is observed as follows: The pause between the first and second sound becomes of the same length as that between the second and first (systole = diastole), and at the same time they equal each other also in character and loudness. The action of the heart is similar to the tick-tack of a watch, and if, as is frequently the case, tachycardia exists at the same time, the whole

[1] See this.
[2] See Preliminary Remarks, p. 167.
[3] See p. [191 f].
[4] See Preliminary Remarks, p. [168 ff].
[5] See section on Heart-murmurs and Pulse.

movement reminds one of the fetal heart-sounds (embryocardia, pendulum-rhythm). Whether with or without tachycardia, the phenomenon is very suspicious of commencing weakness of the heart. We have observed it particularly in the diphtheria-heart and in chronic myocarditis as a forerunner of the gallop rhythm.

Divided or Double Heart-sounds.—These, as we have seen above, are ordinarily without significance if the condition otherwise is one of health [see page 187]. They occur also in pathological conditions, and are then of diagnostic meaning. We bring together here (Fig. 61) the cases in which, instead of two heart-sounds, we hear three, without sharply separating between "divided" and "doubled" sounds.

Division of the second sound at the apex occurs in *mitral stenosis.* It may conceal a diastolic sound, which, with the patient in the up-

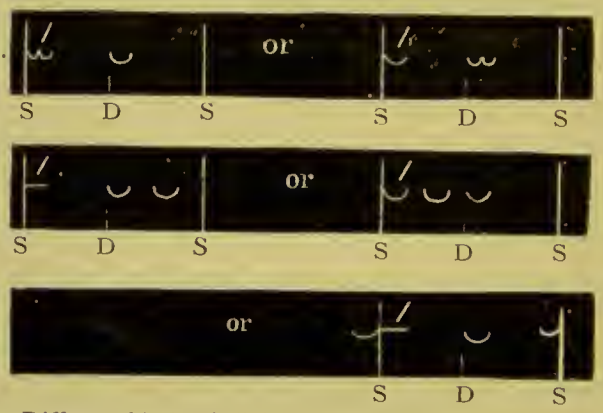


FIG. 61.—Different kinds of division and doubling of the heart-sounds.

right position and heart excited, sometimes can only be distinctly heard by placing the stethoscope at the outer left end of the apex-beat. We may especially refer a divided second sound at the apex, according to my experience, to mitral stenosis, in case there are, besides, undoubted signs of *mitral insufficiency;* and if at the same time the pulse is too small for a compensated mitral insufficiency, an incompensation is thereby excluded.

Further, *a divided second sound* is heard in *pericarditis adhæsiva* and systolic retraction of the apex-beat. (Friedreich's explanation of the phenomenon may be doubted.)

Finally, here belongs the *gallop rhythm*, sometimes :



FIG. 62.—Gallop rhythm.

that is, three similar short ringing sounds, of which either the second or third has an accent, but in many cases neither has an accent. This

gallop rhythm may, but quite exceptionally, be observed in health with excited action (I have seen several cases). It is also observed in emphysema, contracted kidney, arterial sclerosis, heart-disease with slight incompensation. But it generally indicates severe, often fatal, heart-failure, and especially in infectious diseases. It is particularly frequent in children; it may here—for example, in *diphtheria*—be the first sign of beginning paralysis of the heart, even before the pulse becomes markedly quickened. In my opinion the gallop rhythm may be explained in the same way as the divided sound, the ventricles not contracting at the same time. This question will be variously answered by different authors.

Metallic Heart-sounds.—They come from the resonance of a large smooth-walled layer of air close over the heart, as is the case in *pneumopericardium*, not infrequently in *pneumothorax*, and in individual cases of large *cavity in the lung* with smooth walls which lies close to the heart. *Intestinal or peritoneal meteorism*[1] or a very much *inflated stomach* may sometimes cause metallic heart-sounds.

In pneumopericardium, also in cases of inflation of the stomach with gas, if the action of the heart is very strong or excited, the sounds may be so loud that the first, or even the first and second, can be heard at a distance.

Organic Endocardial Heart-murmurs.—By *endocardial heart-murmurs*, as the name implies, we understand murmurs arising within the heart in distinction from those arising in the pericardium. Endocardial murmurs are again distinguished as organic and inorganic according as they are dependent upon anatomical changes or not. We now consider the former.

Organic heart-murmurs are caused by stenosis of the openings, or by imperfect closure of the valves or insufficiency, both the ordinary and the relative insufficiency of the valves. They furnish us with an important means of recognizing the so-called valvular defects.

If fluid is flowing through a tube which suddenly at a certain point is contracted, from this stenosis eddies arise in the current below that point, and these eddies will cause murmurs. If the fluid flows very rapidly, the eddies and their sounds are increased. Normally, the blood passes through the openings of the heart without sound, since there is no notable narrowing of the channel of the blood; but if an *opening is narrowed*, then eddies and sounds are produced, and so much the more markedly if there is compensation, when the blood from the section of the heart lying behind the narrowed opening is driven with much greater rapidity than normal through the narrowed opening.[2]

Such a murmur will be heard at the moment when normally the blood passes through that opening; that is, at the systole if an arterial opening is narrowed; at the diastole if a venous opening is affected (auriculo-ventricular).

But insufficiency of the valves produce murmurs which are to be explained in the following way: The effect of insufficiency is such that the blood, which, in the preceding stage of the heart's action, passes through the affected opening, in the following stage, in which

[1] See both of these.

[2] See Preliminary Remarks, p. 168.

the valves of that orifice would have closed, partly flows back; it likewise flows against the blood normally flowing into the cavity, and rebounds with it; thus eddies arise and also a murmur. The intensity of this murmur depends, in the first place, upon the degree of insufficiency, and, again, very materially varies with the strength of the heart's action; for the greater this is the more marked is the difference in pressure and the more violent the backward current which it causes.

Likewise, there occurs the *murmur of insufficiency* in that stage of the heart's action in which the affected valves ought normally to close; that is, at the arterial openings with the diastole, and at the venous openings with the systole.

Moreover, it appears to me to be unquestionable that, in the great majority of cases of insufficiency, the murmur is increased by the simultaneous occurrence of a murmur from stenosis; for the reflux current of blood certainly flows through a narrowed opening if the insufficiency is not greater than it usually is. I also think that, in connection with this, in cases of severe aortic insufficiency (N. B., with full compensation), we find the diastolic murmur especially soft. (See further regarding this the following, upon the influences that affect the loudness and character of the heart-murmurs.)

Localization of the Murmurs.—The next diagnostic point of importance is that, from the location in the region of the heart where a murmur can be heard most distinctly or where it is loudest, we can determine whence it arises—that is, at which opening the valves are diseased. The auscultation-points already mentioned, empirically found, serve here as points of departure. We listen—

At the apex of the heart—that is to say, at the point of the apex-beat—for the mitral valve, the left venous opening.

Over the lower part of the sternum—for the tricuspid valve, the right venous opening.

In the right second intercostal space, close to the sternum—for the [aortic] opening and the auricular semilunar valves.

In the left second intercostal space, close to the sternum—for the opening [of the pulmonary artery] and the pulmonary semilunar valves.

But it is to be noticed that the murmur caused by aortic insufficiency is, as a rule, not heard in the right second intercostal space, but is most distinct over the sternum, sometimes even in the third or fourth intercostal space at the left of the sternum; since it is also caused by the backward flow of the blood, it is conducted in the direction of the ventricle. Analogously, but only exceptionally, the murmur of insufficiency of the mitral valves may be noticed most markedly, not at the apex, but on the left of the base of the heart; that is the case when the dilated left auricle, with its appendage, lies somewhat forward (Naunyn).

The murmur of stenosis of the left auriculo-ventricular opening is often distinctly heard close to the outer edge of the apex-beat.

Relation of the Heart-murmurs to the Time of Action of the Heart.—It follows from the above discussion that the organic heart-murmurs are very closely connected with certain instants of the action of the heart, and, further, that they are divided into systolic and diastolic. And thus we hear in—

Stenosis of the aorta: A systolic murmur in the right second intercostal space.

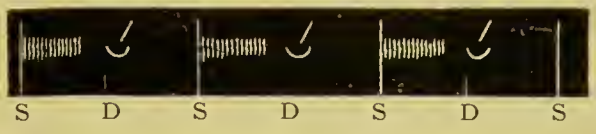


FIG. 63, *a*.[1]

Aortic insufficiency: A diastolic murmur at the same place, or, better, lower down to the left of this, over the sternum.[2]

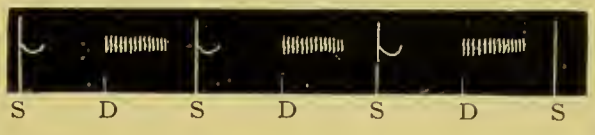


FIG. 63, *b*.

Mitral stenosis: A diastolic murmur at the apex, the first sound valvular; or approximately so, if the second sound is heard at all.[3]

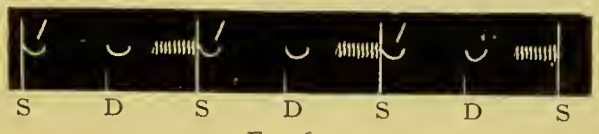
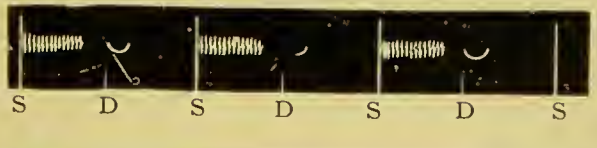


FIG. 63, *c*.

Mitral insufficiency: A systolic murmur at the apex of the heart,



or,

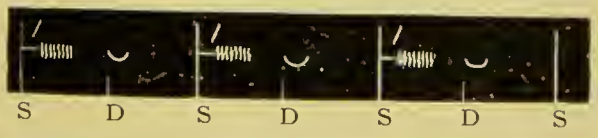


FIG. 63, *d*.

Quite analogously, in *pulmonary stenosis* and *tricuspid insufficiency* we hear a systolic murmur, in *pulmonary insufficiency*, and *tricuspid stenosis* a diastolic murmur at the corresponding points.[4] Of these valvular defects of the right side of the heart the only one frequently present is *tricuspid insufficiency*, and this is relatively much more frequent (in great weakness of the heart) than insufficiency caused by endocarditis. Pulmonary insufficiency and stenosis are almost always congenital, and then are very often associated with a *permanently open foramen ovale*.[5]

Systolic murmurs in stenosis of the aorta and insufficiency of the mitral valve, and the diastolic murmur from aortic insufficiency gener-

[1] [Figs. 63*a*, *b*, *c*, *d*, *e*, *f*, indicate endocardial heart-murmurs.] [2] See above.
[3] See more exactly below. [4] See above. [5] Regarding this, see later.

ally are directly joined with the sound affected by them; but these sounds are thus always weakened, or the sound completely disappears and the murmur takes its place. In such cases the sound may still be heard if we remove the ear a short distance from the ear-plate of the stethoscope. Probably the weakened sound is not to be referred to the valve that is affected, but is conducted so as to be heard elsewhere.

On the other hand, a peculiar condition commonly belongs to the *diastolic murmur of mitral stenosis;* it occurs at the end of the diastole as a so-called *presystolic* murmur, or, if it is present at the beginning of the diastole, it becomes stronger toward the end; hence, either—

or,



FIG. 63, *e*.

The explanation of this remarkable phenomenon is very simple: toward the end of the diastole the auricle contracts and drives the blood with greater rapidity through the narrow ostium venosum; hence the strengthening of the eddy and murmur.

A diastolic aortic murmur may be heard at the apex only as presystolic, and then, if one does not examine exactly for the other evidence, it may be taken for a mitral murmur and be interpreted as a mitral stenosis. However, when there are adhesions of the pericardium we also occasionally hear a presystolic murmur.

If a presystolic murmur is very short, it may make the impression on the ear of a "tone," and the second sound seems divided. In such cases there are difficulties in making a diagnosis.[1]

In most cases a little practice enables one to recognize in what period of the action of the heart an endocardial murmur belongs. But if there remains the slightest doubt whether a murmur is systolic or diastolic, then the examiner must observe the action of the heart by palpating at the same time he is auscultating, and this is best done by applying a finger to the common carotid in the neck; here the pulse is almost simultaneous with the ventricular systole, and hence demonstrates the time of its occurrence.

We cannot employ the radial pulse, because it is felt too long after the systole. When the action of the heart is very irregular, and still more when it is very much accelerated, it is very difficult, or it may be entirely impossible, to distinguish between systole and diastole.

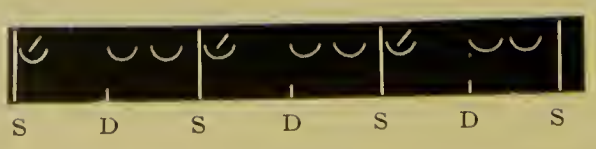


FIG. 63, *f*.

[1] See p. 190.

Loudness of the Endocardial Murmurs.—From what has already been said it is evident that the loudness of the murmur is not alone dependent upon the severity of the valvular lesion. It is also a very great mistake to draw a conclusion about the degree of the stenosis or insufficiency from the loudness of the murmur; regarding this, the effects of the valvular lesions upon the heart and circulation, especially the pulse (which see), are much more determinative.

Murmurs are very much affected by the strength of the action of the heart; they are plainly louder when the heart is excited, and hence when they are indistinct, if the patient is able to do so, and is not harmed by it, he can first bend forward and stretch out a few times, or he can sit up and lie down again several times in bed before we auscultate him. On the contrary, a murmur previously distinct becomes, without exception, more feeble if the strength of the heart declines. In very marked weakness of heart the murmur may even become entirely imperceptible; hence in disease of the heart the murmurs entirely disappear if an unfavorable turn takes place; also, they disappear in cases of heart-disease where the patient is overtaken with a severe febrile disease. Hence, an exact diagnosis of disease of the heart, if the heart is weak, is always uncertain, and often impossible, whenever the action of the heart is accelerated.[1] Hard (calcareous) or rough valves have the *effect of strengthening* or *sharpening the murmurs* of stenosis, or, perhaps, also of insufficiency; also, in individual cases the murmur may be changed by the relaxation or rupture of the tendinous cords of the valves.[2] In other respects the strength of the murmurs is dependent upon the same influences as affect the heart-sounds.[3]

In rare cases the heart-murmur is so marked that it may be heard *at a distance*, without laying the ear over the chest. Such murmurs may sometimes be perceived by the patient. The murmurs which sometimes have this peculiarity are chiefly those which arise at the aortic orifice.

Murmurs differ very much in character: murmurs of insufficiency are, as a rule, softer, blowing, and, indeed, the murmur of *aortic insufficiency* manifests itself often by its length and remarkable delicacy (it may easily be overlooked), while that of *mitral insufficiency* usually is louder, but not quite so long. Of the murmurs of *stenosis*, that of the aorta is generally loud, "sawing;" *mitral stenosis*, on the other hand, is almost always very soft, peculiarly rolling or "flowing," or seeming to consist of several very soft sounds. This murmur is sometimes imperceptible, even with strong action of the heart.

Under some circumstances aortic or mitral murmurs of insufficiency may be musical; that is, they contain a sound which approaches a distinct, always very high musical, tone. In such cases it has frequently been found at the autopsy that the suspected cause of this phenomenon in such cases was a perforation of the semilunar valve, also torn floating shreds of valves, sinewy threads in the lumen of the ventricle, floating torn shreds of papillary muscle, etc. These con-

[1] See Relation of Heart-murmurs to the Time of Action of the Heart, p. 194.
[2] See Character of the Murmurs.
[3] See these.

ditions generally furnish no indication as to the particular heart-lesion; it is, therefore, of no value to recognize them during life. In many cases, moreover, of which two came under my own observation, it happens that at the autopsy nothing is found to explain the occurrence of the musical murmurs during life.

Metallic murmurs occur under the same conditions as metallic heart-sounds,[1] in general if there is a resonant air-space near to the heart.

Murmurs that may be felt: endocardial whizzing, "frémissement cataire," cat's purring. This occurs generally, but by no means always, with murmurs that are distinguished by their loudness. Locally, their most distinct perception by touch always corresponds with the locations where they are heard most distinctly. We palpate with the hand or finger-tips and recognize thus, though only in rare cases, a fine whizzing, which is most like what we feel when we stroke the back of a purring cat.

In this way, by the aid of palpation, we may prove the existence at the apex of systolic and diastolic or presystolic mitral murmurs, and in the right second intercostal space of systolic and diastolic aortic murmurs. Defects of the right heart seldom produce murmurs that can be felt. The palpation of endocardial murmurs has so subordinate a value that we can never permit ourselves to dispense with auscultation, which yields so much sharper and clearer results.

Transmission of Heart-murmurs.—It is understood that an endocardial murmur is very often not confined to that spot on the thorax where it is auscultated, but will be heard at some distance away from it. The conduction takes place especially in the direction of the blood-current. Thus an aortic systolic murmur is often heard even over the carotid in the neck. On the other hand, the diastolic aortic murmurs generally are perceived over the sternum, even louder than in the right second intercostal space; but they are also often to be heard as far down as the apex. Systolic blowing in mitral insufficiency is sometimes conducted toward the right as well as farther upward. On the other hand, diastolic [presystolic] murmur from mitral stenosis is always sharply confined to the left border of the heart. An *inorganic systolic pulmonary murmur* which can be heard some distance downward from the base of the heart very often disturbs or deceives us.

Combination of Several Murmurs.—This results from the *combination of several valvular defects.* It more frequently happens that insufficiency of a valve is connected with stenosis of the opening to which that valve belongs. Then we hear at a particular spot a murmur with each of the two stages of the heart's action. It is more difficult to interpret what is heard when the disease affects different openings or valves, and especially if there are two murmurs, both of which occur with the systole (mitral insufficiency and aortic stenosis), or both in the diastole (mitral stenosis and aortic insufficiency). Then it may happen that only one valve is supposed to be diseased, and that the second murmur which is heard is transmitted from the first. But also a mistake in the opposite direction may be possible—namely, that we assume

[1] See Metallic Heart-sounds, p. 191.

that there is a combination of two valvular affections when in fact there is only one, as when a murmur of aortic insufficiency which is heard at the apex is considered as a new, independent murmur produced by mitral stenosis. The differentiation by auscultation is made in two ways: 1. By the *character of the murmur.* If one is blowing and the other is rough, there certainly are two murmurs; if both are alike, then there may be only one, which is conveyed from the opening where it arises to a second opening. Yet it might be that even in this case there were two murmurs, with different origin. 2. We auscultate step by step from the point where we can hear one to where the other exists, as from the apex to the aorta. If the murmur is everywhere distinct, only that toward one spot it gradually becomes louder, then it arises at this point and is conveyed to another. But if it is lost somewhere on the way from the apex to the aorta, and is again heard at the aorta, then there are two murmurs.

This procedure may answer the purpose, but it often fails, and in such difficult cases auscultation alone cannot decide, but we must take a view of the whole picture of the heart and vessels in order to reach a diagnosis.[1]

Finally, *murmurs* that arise *in the neighborhood of the heart* may be mistaken for heart-murmurs. Those that come from the trachea and bronchi can easily be excluded by having the patient, if necessary, hold the breath. But it is more difficult to discriminate between heart-murmurs and those that have their origin in the aorta (especially aneurysm).[2]

Inorganic, Anemic Murmurs.[3]—These are so designated because they occur in all forms of *anemia*, both slight and severe, but especially in *chlorosis*, in all *wasting diseases*, and also in *febrile diseases*, without there being any disease of the heart or vessels. They serve as a sign of anemia; they generally entirely disappear with the removal of this condition.

In very pronounced cases there are very *soft*, *systolic*, *blowing murmurs*, which are heard over the pulmonary artery or lower down with indefinite location, or they may even be heard over the apex. But not very infrequently such an inorganic murmur is also sharp, even very loud; on the other hand, it is very seldom diastolic; also we may almost say that it never is heard over the aorta. Thus the other signs of valvular disease are wanting, especially hypertrophy of a ventricle, while the pulse gives evidence of anemia, and there are murmurs in certain vessels, especially the veins of the neck.

Sometimes there is at the same time considerable dilatation of the heart, as takes place in anemia;[4] on the other hand, we have those marked dilatations which give rise to murmurs from *relative valvular insufficiency*, and which may also exist in severe conditions with which we are not at present concerned.

It is very difficult to explain anemic heart-murmurs. Nothing of what has already been said regarding murmurs seems to us to be applicable here: we think, with others, that the nature of the phenomena differs in different cases, and in many cases we may apply Sahli's

[1] For further on this, see below.
[2] For further on this, see below.
[3] Synonyms: Accidental Blood-murmurs.
[4] See above.

supposition that venous murmurs from the large veins in the thorax lie behind these heart-murmurs.

For distinguishing them from the organic heart-murmurs it is in the first place necessary to call to mind what has been mentioned as characteristic of anemic murmurs, and then to observe whether there are other signs of anemia present. Further, a valvular defect is to be excluded by the most careful examination of the heart and pulse. It is true that in many cases the phenomena are such that we can only obtain a clear idea by long observation, especially remarking whether treatment of the anemia removes the murmur. It is very difficult to decide that a diastolic murmur is due to anemia.

The author recalls having seen two cases of pronounced pernicious anemia complicated with mitral endocarditis and mitral insufficiency, in both of which the differential diagnosis between anemic murmurs and the valvular disease mentioned could not be positively established during life. In both there existed simultaneously considerable emphysema which concealed the slight hypertrophy of the left and right ventricles.

Pericardial Murmurs [Friction-sounds].—The name explains the situation of these murmurs. Their nature is the same as pleuritic friction-sounds; they are caused by the friction of the visceral and parietal pericardium made by the action of the heart when their opposing surfaces rub against one another; they do this when the surfaces are rough, exceptionally even if they are simply unusually dry.

The relation of the friction-sound to the action of the heart is of great importance: it occurs, not in close conjunction with the sounds, but between them, either only during the systole or more frequently in both stages, but generally louder with the first sound:



FIG. 64, *a*.[1]

More rarely, tolerably closely before and after the second sound:



FIG. 64, *b*.

or covering the first sound:



FIG. 64, *c*.

[1] [Figs. 64, *a*, *b*, and *c* indicate pericardial heart-murmurs.]

Less important than the preceding is it to note that we have near to the ear a ringing, short scratching, scraping, shuffling, more rarely a creaking, sound, one which with a little practice is generally easily correctly recognized by its acoustic character. It is generally very sharply defined as to location, and is most frequently heard at the base of the heart, but often farther down at the left of the sternum.

The rubbing of marked pericardial friction-sounds can be felt by applying the hand to the spot. Several special peculiarities of these friction-sounds will be mentioned when we treat of Differential Diagnosis.

Pericardial friction-sounds occur:

In *pericarditis*, when the surfaces of the pericardium, where the fibrinous exudation exists, rub against each other without becoming adherent. Hence, we hear friction-sounds in *pericarditis sicca* so long as it is not adhesive, and in *pericarditis exudativa* if there is fibrinous exudation without enough fluid completely to keep the surfaces of the pericardium apart. This is why the friction-sound is generally heard at the base of the heart or near to it; it is not infrequently heard there as the first sign, and then often disappears as the exudation increases, and it may again return when the exudation diminishes. The *disappearance* of a previously existing pericardial friction-sound may depend upon one of four causes: 1. The complete decline of a pericarditis without any sequelæ. 2. By the addition of a fluid exudation. 3. By adhesion of the pericardial surfaces. 4. From great weakness of the heart. It is necessary to ascertain in every case which of these four causes is operating. If there is no evidence of the second or the fourth, then the first and third must be considered; and between these it is possible to make a differential diagnosis only in very rare cases.

They also occur in rare cases of *tuberculosis of the pericardium* (which usually results in adhesion), quite exceptionally with fragments of fibrinous cords and calcifications in the pericardium, and in *abnormal dryness of the pericardium*, as in cholera.

The differential diagnosis between pericardial and endocardial murmurs is generally very easy for those who are accustomed to hear both sounds, frequently by the character of the pericardial sounds and the circumstance that they sound so near the ear. Musical persons generally also immediately recognize the difference in time.[1] But the following may enable us to distinguish between them:

(*a*) Very much the most important is the consideration of the whole picture of the disease (form of the dulness, apex-beat, sounds, pulse, etc.).

(*b*) *Change of Position.*—The pericardial sound almost always changes, and much more than the endocardial, with change of position.

(*c*) Strong pressure with the stethoscope. If we press exactly at the right spot, especially if it be in an intercostal space, sometimes the pressure very strikingly increases a pericardial sound, but never an endocardial one. But in the majority of cases, even of the former, the sounds are not increased by pressure; hence it is merely confirmatory when it exists, but failure to notice it has no meaning.

(*d*) Pericardial sounds often change their location, strength, and

[1] See above.

character in a few hours; they may even very quickly disappear and very suddenly return.[1] Endocardial murmurs are markedly chronic and regular. Very exceptionally they come and disappear suddenly if they are organic, and only in exceptional cases when due to heart-weakness.

Extra-pericardial Friction-sounds.—The friction-sounds which are heard close to the heart, and even over it, and which resemble them in sound, may be very easily confounded with the pericardial sounds. This extra-pericardial sound is, in the great majority of cases, a *pleuritic friction-sound* which is caused by the contact of the pleura with the heart, especially at the lingula, and which by the mechanical effect of the action of the heart results in thrusts which correspond with the movements of the heart. It is distinguished from pericardial friction-sound in that it is greatly influenced by the breathing: it is often heard only with deep inspiration or, on the contrary, during very superficial breathing. In individual cases we hear it as pleuritic friction with strong breathing, while with quiet breathing it has the time of pericardial friction-sound.

There occurs also a peritoneal friction with peritonitis, involving the lower surface of the diaphragm (subphrenic peritonitis), and quite exceptionally over the liver. This sound is transmitted by the motion of the heart upon the diaphragm as a pseudo-pericardial sound.

The *differential diagnosis* of these sounds from pericarditis will depend upon the other signs of a pleurisy or peritonitis, and with reference to pleuro-pericardial friction the effect of the breathing is to be considered. Hence the differential diagnosis may here be very difficult, because sometimes a pleurisy close to the heart may by contiguity awaken a pericarditis.

Fine crepitations, like those in emphysema of the skin,[2] occur in the neighborhood of the heart, synchronous with the action of the heart, in mediastinal emphysema.

Metallic pericardial splashing results from fluid and air in the pericardium (pyopneumocardium), exactly as we have succussion-sound with hydropneumothorax, only that the succussion is caused by the heart itself. Moreover, after the analogy of extra-pericardial friction-sound, a pseudo-pericardial—in fact, pleuritic—splashing, simultaneous with the motions of the heart, occurs with hydropneumothorax, where the motions of the heart are communicated to the fluid. This happens exceptionally, too, with large cavities which lie close to the heart. Finally, it happens that the movements of the heart produce metallic resonance or splashing sounds in the stomach. This has been observed exceptionally and temporarily in healthy people if the stomach was very full. As a permanent phenomenon it has been described in isolated cases of coalescence of the pericardium (Riess). Here it is probable that the coalescence of the heart with the pericardium, and also the eventual adhesion of the pericardium to the upper surface of the diaphragm, may produce a more direct transmission of the beats of the heart to the wall of the stomach, or, in turn, to the contents of the stomach.

Exploratory puncture of the heart is only to be undertaken with

[1] See above.

[2] See p. 49.

reference to the performance of puncture, and hence belongs under therapeutics.

EXAMINATION OF THE ARTERIES.

Usually we select the *radial pulse*, which, because of its importance, requires a separate and complete consideration. Then we can add to this the description of the characters of the other arteries.

I. The Pulse, its Palpation and Graphic Representation.

From the commencement of medical study the radial artery has been examined where it passes between the styloid process of the radius and the tendons of the long flexors of the hand and fingers. The examination of the pulse is not a simple thing. It requires much practice, and hence it is the more important, in order to be able to recognize the differences and peculiarities of different cases, always to take the pulse at the same artery; but it is easy to understand that the radial artery is preferable because of its location, and hence it has been selected.

At the same time, it is to be noted that the arteries of the forearm not infrequently pursue an abnormal course. The most frequent anomaly is of the radial artery, in that it passes across the radius outward and upward; or the ulnar artery may be enlarged at the expense of the radial. In the latter case, of course, the pulse of the radial is small. These anomalies may be on one or both sides.

Arterial sclerosis usually influences the examination of the pulse to a very marked degree, and must therefore not be overlooked.

Palpation of the Pulse.—The arm being held in an unconstrained position, we palpate the radial by making slight pressure upon it with the tips of the first and second fingers. Generally the impression is threefold: we learn the *condition of the artery itself*, the general state of its *fulness with blood*, and its *pulsatory dilatation and contraction*. This latter constitutes the pulse in its narrow sense.

We study the pulse with reference to its *frequency*, its *rhythm* (whether the succession of beats is regular or not), and its *quality*. First we consider the normal pulse; then the pathological departures from it with reference to these three points of view.

1. The Normal Pulse.—Its *frequency* varies with the *period of life*. In the newly-born it varies much: when active it is as high as 140 in the minute, but during sleep it is 90 to 100. Up to the tenth year it is about 90, and at about the sixteenth year it is 76 to 70. It remains at about this number in healthy persons till old age, when it sometimes increases again to about 80 in the minute.

There are variations, it is true, from these figures in perfectly healthy adults, who may continuously and regularly have a lower pulse, even down to 60 (or still lower). *Sex* makes a slight difference, the female average being a few beats more than the male at the same age. Moreover, the *size of the body* has some influence: the average of large persons is somewhat less than that of smaller persons, *cæteris paribus*.

The *daily variations* in the frequency of the pulse correspond with those of the bodily temperature; the maximum is generally between

noon and evening, the minimum in the early morning; the difference is generally less than ten, seldom more than twenty, beats.

Of about the same value is the variation of the pulse with reference to the *position of the body:* its frequency is highest in standing, less while sitting, and least while lying down. It varies also with the *external temperature* in case the latter changes considerably from the average: the lower the temperature the higher the pulse.

Meals, especially of food that is rich and of hot dishes and drinks, quicken the pulse for one or two hours. *Sleep* has no essential effect, though the pulse rises, and generally considerably for a short time, at the moment of waking, even when this is without noticeable excitement.

Movement of the body always increases the frequency, under some circumstances even till the frequency is doubled. Active *deep breathing* increases it. *Mental excitement* of any kind, as fright, anxiety, joy, joyful or painful tension, likewise quickens the pulse, but very differently in amount in different individuals according to their general excitability.

All the above-mentioned influences manifest themselves with very marked variations according to the bodily constitution and the character of the nervous system [temperament]. Pale, delicate persons, who are also excitable, show the greatest increase in frequency. During convalescence merely rising in bed, a little food, joyful or sad news, considerably quickens the pulse. In disease this is still more the case.

Method of Observing the Pulse.—After excluding the temporary influences that have been mentioned, we count by the second-hand of the watch for twenty seconds; where greater exactness is required, for a half or full minute. Sometimes in hospitals the nurses employ small sand-glasses; of course their accuracy must be carefully tested. [In England and America these glasses are not used.] Sometimes in sickness the pulse is so frequent that it cannot be counted. It has been recommended, under these circumstances, to try to count every other beat, and then to double the result. If the radial pulse cannot be felt, or if we suspect that some beats drop out,[1] we can then count while we auscultate the heart.

In connection with the employment of temperature-charts we have become accustomed to note upon the chart, every time the temperature is taken, also the frequency of the pulse and respiration; thus we obtain upon the fever-chart a continuous line representing the pulse, which materially aids in forming a judgment of it. (Regarding the value of this continued observation of the pulse, see below.)

The *rhythm* of the pulse in perfect mental quiet and during quiet breathing is in health regular. But mental excitement easily makes the pulse somewhat irregular, especially in nervous persons. Again, the rhythm of the pulse is changed with many persons during deep breathing, especially, too, in nervous persons. Usually at the end of expiration and the beginning of inspiration it is quicker, while at the height of inspiration and the beginning of expiration it is slower.

Normally the pulse at the two radials is exactly simultaneous; the

[1] See under Intermittent Pulse.

crural pulse is also approximatively simultaneous with the radial. But if we compare the radial with the action of the heart, we notice that it is always notably later than the corresponding systole.

Regarding the *quality* of the pulse: the radial in health has a certain general fulness and hardness, and the separate pulse-waves also have a certain size, hardness, and form. All these peculiarities exhibit not inconsiderable variations within the normal. Correct estimate of them by palpation is a matter of much careful practice.[1] Here it is next to be remarked that in the normal pulse *equality of its separate beats* is desirable (*equal pulse*); only quite small, scarcely perceptible inequality sometimes occurs, again especially with nervous persons. A general symmetrical increase in the hardness of the pulse and enlargement of its waves are results of physical exertion, mental excitement, etc.—in short, from anything that temporarily quickens the action of the heart.[2]

2. Pathological Frequency of the Pulse.—We distinguish a *pulsus rarus* (slow, infrequent pulse) and a *frequent pulse* (accelerated pulse).

Care must be taken not to confound *pulsus rarus* with *pulsus intermittens*, in which the pulse-waves are unequal, and some of them so weak that they cannot be felt at all. With some practice *pulsus intermittens* is usually easily recognized. In some cases, however, even a more practised physician may once in a while be mistaken, as when the inequality of the heart-beat, and hence the intermittence, is periodic, so that the pauses between the palpable pulses become nearly equal. Here belong particularly *pulsus bigeminus* and *trigeminus alternans*.

In case of doubt auscultation of the heart always at once removes the doubt.

Of late *pulsus rarus* has frequently been an object of scientific investigation (Grob, Riegel). Occasion has even been taken to distinguish it by a particular proof of respect: it has received a name. Retardation of the pulse, so that the number falls below 60, is called *bradycardia*. This name may be confounded by the hearer with the opposite, *tachycardia*, unless the speaker pronounces very distinctly.

As a physiological phenomenon *pulsus rarus* appears only occasionally; at least it is extremely rare in the perfectly healthy. In persons beyond the thirties it always gives rise to a suspicion of coronary sclerosis or of fatty heart, if other phenomena are entirely absent.

The transition condition to the pathological *pulsus rarus* is that in which it occurs during the puerperium and in the state of starvation.

Pathological *bradycardia* is found under very different conditions. The cause is partly direct or reflex irritation of the vagus center, probably also irritation of the vagus trunk; partly increase of arterial pressure by vaso-motor or purely muscular contraction of the peripheral arteries; partly diminution of pressure by loss of blood; then indirect influences on the heart-muscle of substances circulating in the blood also come into consideration; and, finally, anatomical changes of the muscular tissue of the heart or of the endocardium.

Retardation of the pulse is therefore observed—

1. In individual cases of pathological increase of the work of the heart—namely, in *acute nephritis*, especially the *nephritis of scarlet*

[1] For particulars regarding the different forms of pulse, see p. 208, *f.* [2] See this.

fever. Hypertrophy of the left ventricle is often included here. But the diminished frequency of the pulse is very slight.

2. In the opposite condition of diminished pressure in the arterial system in consequence of *hemorrhage*, and in individual cases of febrile diseases ending in fatal collapse.

3. Sometimes with *stenosis ostii aortæ;* here the difference is usually very slight—about 60 beats.

4. In *disease of the heart-muscle*, especially in *fatty heart*, but also in *fibroid myocarditis* (but here we must be on our guard not to confound it with intermittent pulse);[1] 48 to 36 beats are here not at all infrequently met with. The lowest number pretended to have been observed is 8.

In acute dilatation of the heart from over-exertion, whether it occurs in a diseased heart or one previously sound.

5. In *old age*, sometimes without any gross disease of the heart, and in *marked inanition* (from stenosis of esophagus, pylorus, etc.). Here, also, the slowing of the pulse may be considerable—even to 48 or less.

6. In *disease of the brain* or *of the meninges*, which results in irritation of the vagus center. This may really be only mechanical, from increased intracranial pressure (*tumors, hemorrhages, hydrocephalus*) or from inflammatory irritation (*acute meningitis*, especially basilar). The slowing is considerable in the majority of cases.

7. In individual rare cases of irritation of the vagus nerve by compression (tumors) or by inflammation (abscess) in its neighborhood.

8. In neurasthenia, hysteria (rare).

9. In all possible, mostly painful, diseases of the abdominal organs, especially in ulcer of the stomach.[2]

10. In the *critical decline of fever* in acute febrile diseases, possibly from the effect of certain products of the fever upon the heart or the vagus center, an effect which is only manifest when the quickening effect of the high temperature upon the pulse is past.[3] It is a considerable, but quite temporary, slowing.

11. In *hepatogenic icterus*, from the effect upon the heart of the gall-acids circulating in the blood. The pulse is diminished quite frequently as low as to 48, sometimes still lower. The slowing disappears, and is even followed by acceleration of the pulse, in persistent icterus if there develops cachexia, particularly a decrease of heart-power.

12. In *certain intoxications*, especially in lead and in acute alcoholic poisoning.

Bradycardia occurs in a pronounced degree, but intermittently, in colic, particularly in lead colic, also in attacks of pain of other kinds, especially neuralgias; finally, in angina pectoris, not only in the so-called organic angina, notably in coronary angina (angina from sclerosis of the coronary artery), but also the nervous anginas called vaso-motor because produced by a sudden narrowing of the peripheral arteries. However, great retardation does not occur in the latter condition, but only in *angina pectoris organica*, especially *coronaria.*

Frequent pulse, or *tachycardia*, occurs—

1. *In fever*, as its chief manifestation. We recognize a general

[1] See this. [2] Compare bradycardia in colic. See below. [3] See below.

relation between the elevation of the temperature and quickening of the pulse—to every degree of heat above 37° the pulse increases 8 beats above the normal (Liebermeister); but there are very great

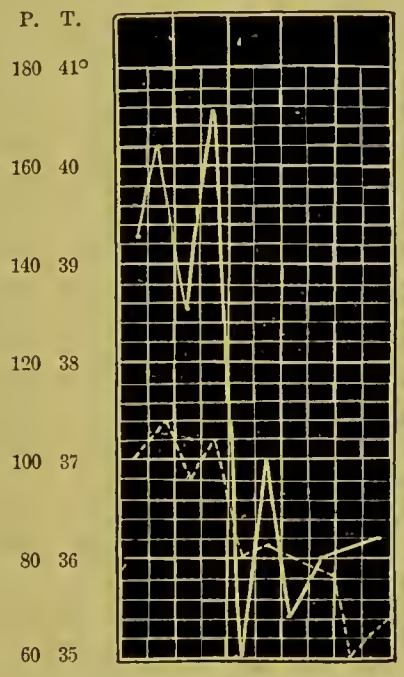


FIG. 65.—Diminution of frequency of pulse after critical fall of temperature in pneumonia. The unbroken line represents the temperature-curve, the broken one the pulse-curve.

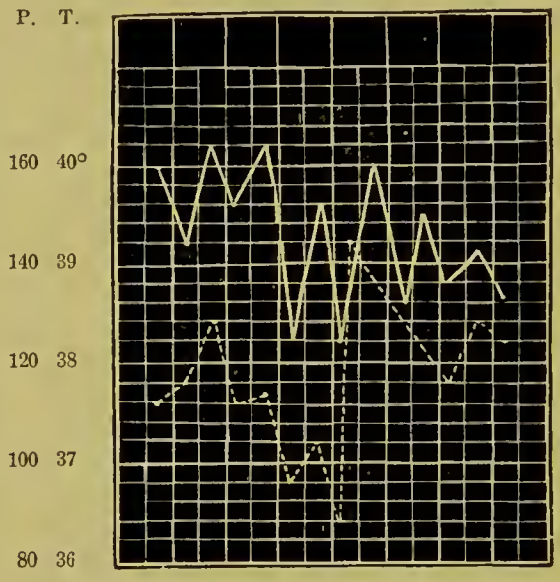


FIG. 66.—Abdominal typhus in the third to the fourth week. The rise in the pulse corresponds with the beginning of pneumonia.

variations from this proportion according to the kind of febrile disease, its localization in particular organs, and, further, with the age of the patient and the strength of the heart. Thus, in *abdominal typhus* [typhoid fever], so long as it is not accompanied by complications, there is only a moderate quickening of the pulse; hence, in this disease a higher pulse—a pulse of 120, for instance—has a graver meaning than, for example, it has in *pneumonia*. This moderate quickening of the pulse peculiar to typhus abdominalis [typhoid fever] is even an aid in diagnosis in severe cases, as distinguishing it from *acute miliary tuberculosis* and *pyemia*, where the pulse is high. It has already been mentioned that in *meningitis* there is slowing of the pulse; when meningitis is added to a febrile disease, it may lower the pulse, previously quickened, to the normal, or may even bring it below the normal. On the other hand, during an *abdominal typhus* [typhoid fever] the addition of a complicating *pneumonia* will, under some circumstances, be first noticed by the increased frequency of the pulse (see Fig. 66). Febrile diseases with *complicating heart-disease* usually have a quicker pulse than the same diseases when the heart is normal. With children the pulse is always very much higher in febrile diseases than with adults.

In the course of febrile diseases the constant observation of the frequency of the pulse is of the greatest importance for estimating the strength of the heart, and with it the general vigor, or showing the

occurrence of complications, etc.[1] It is also to be observed that in

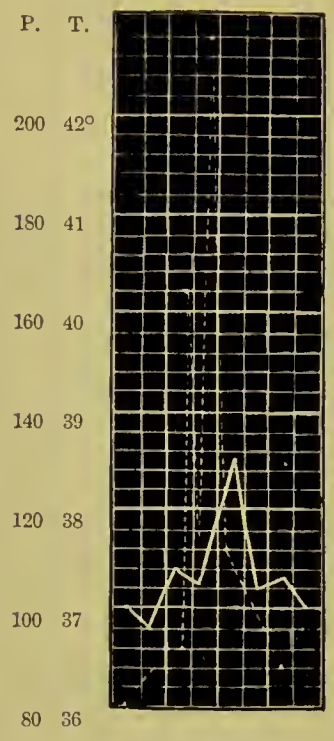


FIG. 67.—Very rapid action of the heart (mitral insufficiency).

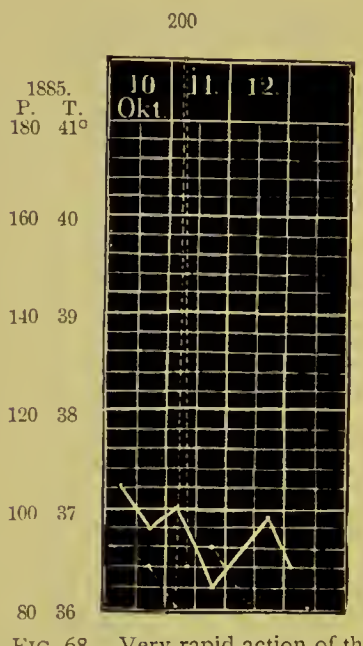


FIG. 68.—Very rapid action of the heart (convalescence from typhus; suspicion of mitral insufficiency).

fever the frequency of the pulse is immediately increased by the least exertion or by excitement.

In general, it is an unfavorable sign when adults have a pulse of

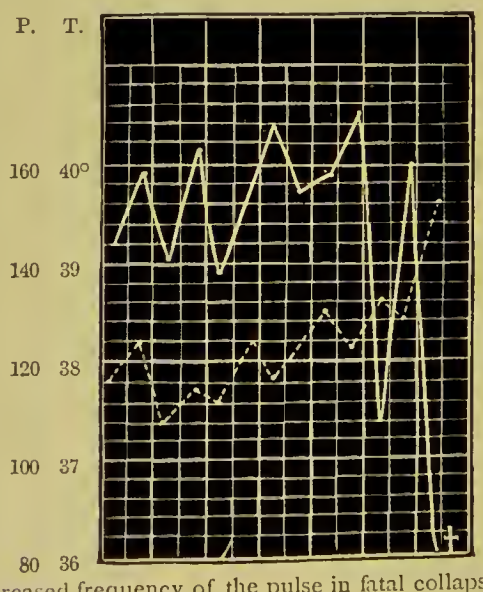


FIG. 69.—Increased frequency of the pulse in fatal collapse (erysipelas).

over 120, and the case requires special consideration. But when it reaches 140 it is a grave symptom.

[1] On this point, see below.

Frequent pulse also occurs—

2. In different forms of anemia, especially in chlorosis.

3. In *valvular disease of the heart* (except only in stenosis of the aorta),[1] and also even with complete compensation.

4. In *heart-failure or paralysis.* Thus, in the collapse of febrile diseases (see Fig. 69), where there is a simultaneous fall of the temperature and rise of the pulse; moreover in the arrested compensation of heart-disease, and in weakening of the heart in consequence of disease of the substance of the heart—more rarely in those cases of heart-weakness which complicate an attack of angina pectoris in organic disease of the heart.

5. With central and peripheral paralysis of the vagus.

6. In *certain neuroses:* Basedow's disease, traumatic neurosis, nervous palpitation, functional (nervous, hysterical) angina pectoris, without the nature of this phenomenon being clear.

7. In any condition of *anxiety*, and with *severe pain.*

Here we have mostly to do with a frequency of the pulse which develops more or less gradually, lasts a certain time, and disappears again gradually. But in a part of the cases mentioned above tachycardia, and that sometimes of an extremely high degree, comes on in attacks—*paroxysmal tachycardia, tachycardic fit.*[2] Such attacks are seen in the conditions mentioned under 3, 5, and 6. It is remarkable that in these attacks there may be missing not only signs of defective motive-power of the heart, but also subjective complaints, even if the attacks occur in persons with organically diseased hearts; for instance, with valvular defects.

In some of the conditions named above—*i. e.* in anemia, in functional neuroses, and in slight diseases of the heart—it may happen that the frequency of the pulse is normal or only a little increased during rest, but is much increased in moderate exertion of the body.

3. Want of Rhythm of the Pulse.—Instead of the normal equal succession of the beats, there may be complete irregularity (*arhythm*); in the most marked degree this is so in *mitral stenosis,* even when there is perfect compensation. Moderate or marked arhythm is very frequent in *myocarditis* (sometimes the inequality of the pulse is here *the only sign*). It occurs during the stage of incompensation in all cases of heart-defect, and sometimes in all forms of marked heart-weakness. Moreover, the inequality of the pulse[3] [irregularity of volume] is more important in judging of the weakness of the heart than arhythm.

If in such arhythm there are individual pauses in which no pulse is felt, then we speak of "suspended" pulse, which may be *pulsus deficiens* —that is, the pauses indicate real pauses in the action of the heart; or it may be a *pulsus intermittens:* these pauses result from weak contractions of the heart, which cannot be felt as far as the radial. We determine, in a given case, which of the two kinds of pulse it is by auscultating the heart.

But there are other forms of irregularity of pulse in which the irregularity of the beats follows a rule: *pulsus bigeminus, p. trigeminus* (where two or three beats are regular and then follows a longer

[1] See above. [2] Compare Figs. 67 and 68. [3] See this below.

pause). These forms generally indicate moderate weakness of the heart.

Lastly, we must mention an especially frequent form of irregularity which stands somewhat between the two last-named forms and complete irregularity—the *pulsus intercidens:* after several perfectly regular beats, suddenly there is one that follows immediately after the last regular one (which is also always weaker), then there generally follows a slight pause. Most frequently it indicates considerable weakness of heart, and is often the forerunner of severe heart-weakness. It occurs in valvular disease and myocarditis.

In order to determine the succession of pulse-beats it is sometimes useful to employ the graphic method.[1]

4. Quality of the Pulse.—As has been already mentioned above, a correct judgment of the size and tension of the radial artery and of the size and form of the individual waves can only be attained by much practice. It is indispensably necessary that there should be acuteness of feeling in the examining finger, much experience of what is normal and what is pathological, and of the boundaries between the two, which cannot be sharply defined in words. The inequality of the examination must be taken into consideration, as it is affected by somewhat individual differences of the location of the arteries, the difference in the subcutaneous fat, or as affected by arterial sclerosis. The exact examination of the pulse may not be possible on account of the abnormal course of the radial artery—the most frequent variation being where the artery winds around the radius to its dorsal surface above the styloid process.

We distinguish the different forms of pulse according to the following points of view:

1. According to the *size of the pulse:* full or empty pulse, *pulsus plenus—vacuus;* a not very clear method of designation. It would be much more suitable to describe the average fulness of the artery, or, still better, its thickness at the moment of its systole—that is, in the depression between two pulse-waves. In this sense the pulse is full in almost all those cases in which it is large,[2] in so far as it depends upon work of the heart, which is strong or increased. But it further depends, to a certain extent, upon the amount of blood in the system; a certain fulness of the pulse, which in a strong person is not remarkable, in an anemic subject indicates a pathological increase in the work of the heart. Within certain limits, moreover, the difference in the fulness of the pulse is individual, being simply dependent upon the internal diameter of the arteries. We are not to confound a full pulse with a case where there is thickening of the wall of the artery by arterial sclerosis.

Large and small pulse: pulsus magnus—parvus. When the *work* of the heart is simply increased, and still more when there is *hypertrophy of the left ventricle*, the pulse is large. There is an exception to this when we have the two valvular defects in which the left ventricle, notwithstanding its hypertrophy, is able to force only a moderate quantity of blood into the aorta—aortic stenosis[3] and mitral insufficiency. The reason for the former is clear; the explanation of the

[1] See this. [2] See below. [3] See Pulsus Tardus.

latter is, that with every systole a part of the blood contained in the left ventricle flows back into the left auricle.

Small pulse depends upon diminished work of the heart, upon an obstruction between the heart and the aortic system (aortic stenosis, aneurysm), and upon marked anemia. It is present in the highest degree in *mitral stenosis*, since in this condition the left ventricle contains an abnormally small quantity of blood, and hence it can drive but little into the aorta.

If the pulse is very small, and at the same time very empty, it is called thread-like or filiform. The trembling pulse (*pulsus tremulus*) is caused by a moderately full artery, in which the wave is imperceptibly small. Both are noticed when the heart is very weak.

Regular and irregular pulse [as to volume]: *pulsus æqualis—inæqualis.* As was previously stated, there occur in health insignificant irregularities in the individual pulse-waves. A very marked inequality is a most important sign of *weak heart*, more important than the irregularity which almost always accompanies it. Only in *mitral stenosis* we have a very markedly unequal (and irregular) pulse without the heart being really weak.

Often, too, there exists in a measure a condition between inequality and irregularity, as follows: A pulse follows the previous one with a shorter pause, then after a longer pause there is one with a stronger beat. Especially in *pulsus intercidens*[1] the between-beat that immediately follows a pulse-wave is always small.

Pulsus alternans is so called when a larger wave alternates with a smaller one. At the same time it is generally bigeminous.[2]

We call a pulse *pulsus paradoxus* which has the peculiarity that in deep breathing, toward the end of inspiration, it becomes weaker or is once or more times omitted. It is an important sign of *pericarditis adhæsiva* with fibroid mediastino-pericarditis, and it arises from the bending or traction of large arterial branches as the thorax is broadened in the act of inspiration and the diaphragm is pressed down.

2. We distinguish the *form of the pulse-wave* as quick or slow, *pulsus celer—tardus.* Here also belongs the *pulsus dicrotus.*

In the *quick pulse* the artery quickly enlarges, and immediately becomes narrow with a like quick contraction. But with a *slow pulse* the enlargement and contraction are slower than normal, and the artery also lingers in the diastole during a portion of time which a trained finger may recognize. With the quick pulse the examiner notices that the stroke is very short, while in the latter it is more a pressure in the vessel against the palpating finger.

Every *pulsus magnus* may exhibit a moderate celerity. Only in *aortic insufficiency* is the pulse decidedly quick. It is a miniature picture of the large fluctuations of pressure in the aorta which quickly follow one another, as with every systole it receives from the dilated and hypertrophied left ventricle an abnormally large quantity of blood, which it immediately disposes of in two directions—sending part back again into the ventricle, and part forward into the body.

It is remarkable that also in heart-weakness there is sometimes a light, quick pulse. It is true that it is always very easy to compress

[1] See above, p. 208.

[2] See this, p. 207 *f.*

it, and between the pulse-waves the walls of the artery fall together very decidedly (*pulsus vacuus*, and at the same time *celer*).

Pulsus tardus is an especial peculiarity of *aortic stenosis*, and at the same time it is generally smaller than normal. How much it may be diminished in size depends upon the degree of stenosis and the strength of the heart. *Pulsus tardus* occurs also with *arterial sclerosis*, likewise with *lead colic*, but also sometimes with other colics, as well as in *peritonitis*.

Pulsus dicrotus will be more exactly described with the Sphygmography of the Pulse.[1]

3. According to the *hardness of the pulse* (tension of the arterial wall) we distinguish hard or tense and soft pulse, *pulsus durus* (*tensus*) —*mollis*. Here we must especially guard against confounding it with arterial sclerosis, which imparts to the wall of the vessel a hardness which has nothing to do with its tension.

We test the hardness of the pulse by endeavoring to compress it with the finger. *It is easy to compress a soft pulse.*

Again, it is really the power of the heart that produces these peculiarities, as well as the active tension of the wall of the vessel. In heart-weakness the small pulse is also always a soft pulse; the large pulse is likewise often hard. With *pulsus tardus* there is almost always a strong action of the heart, and if the heart is hypertrophied, the pulse at the same time is often hard. When the pulse is quick there are constantly marked variations in its hardness.

The hardness of the pulse is especially characteristic in *contracted kidney* with hypertrophy of the heart, also in *lead colic* ("wire pulse"). The pulse is tense also in *apoplexia cerebri* and in commencing *meningitis*, no doubt from irritation of the vaso-motor center.

V. Basch has constructed a so-called sphygmomanometer, which is very useful for measuring exactly the tension in the arterial wall, and thus the blood-pressure. It has been brought out again lately, altered in construction.[2] We omit a description of the apparatus and its mode of use, since each instrument is furnished with directions for using. We only remark that, in our opinion, it should be exclusively applied on the arteria temporalis, because here alone the conditions of the experiment are somewhat equal. And even here the apparatus very often indicates too low a blood-pressure. Normally, the pressure is 80 to 110 mm. of mercury, and the range of values indicated by the instrument in healthy persons is much greater than corresponds to the variations of the arterial pressure. For this reason the instrument does not seem to be adapted for ascertaining the absolute height of pressure. But it is very practical for ascertaining the variations of pressure by continual observation on one and the same patient, if care is taken always, as far as possible, to make the conditions of the experiment equal. Of these the most important is to mark with color or a light line of nitrate of silver the exact portion of the temporalis on which is placed the so-called pulse-cap.

5. Symmetry of the Radial Pulse.[3]—As has been already mentioned, apart from anatomical variations of the artery upon one side, the pulse upon the two sides is perfectly alike as to time and quality.

[1] See p. 214. [2] G. Lufft, Eberhardtstrasse, Stuttgart. [3] Compare p. 216.

It may be disturbed, even to complete absence of the pulse upon one side. Ewald has found that the difference between the two radial pulses under some circumstances is made more distinct by raising the arm. Inequality is caused—

1. By *surgical diseases of the arm*, as fracture of the bone, injuries or operations which displace the radial or which result in narrowing, compression, or cicatricial contraction of the radial, brachial, or axillary artery: in which case the pulse upon that side is found to be smaller.

2. By *tumors of the chest-cavity, of the supra- or infraclavicular fossa*, or *of the axilla*, which press upon the innominate, subclavian, or axillary artery of one side. They weaken the radial pulse even to complete obliteration.

3. By *aneurysm of the aorta, innominate*,[1] also by aneurysm of the subclavian, axillary, and brachial (all very rare).[2]

4. By *emboli and autocthonous thrombi* toward the center from the location of the pulse. In this case the pulse is commonly entirely wanting.

5. In *pneumothorax, also large pleuritic exudation* with compression and distortion of the subclavian. Sometimes the pulse upon the affected side is smaller, also frequently later.

Sphygmography of the Radial Pulse.

K. Vierordt originated the idea of sphygmography. With continued improvements of the apparatus the idea has been further developed by Marey, Wolff, Landois, Sommerbrodt, Riegel [and others].

Sommerbrodt's sphygmograph is the one now most generally used, but it has defects. Recently Ludwig has very decidedly improved upon Marey's instrument, as it seems to me. It can be obtained from Petzold, instrument-maker in Leipzig. [The instrument devised by Dr. Richardson of London is, in the opinion of the Translator, the most practically useful one yet brought out.]

I am very much pleased with the sphygmograph of Jaquet (of Basle), which has a rest for the arm and a mechanism for measuring the time, and a twofold velocity. It fulfils every requisition that can reasonably be made with reference to sphygmography when the instrument is applied to the artery in its normal condition. The instrument can be very highly recommended.

By others the sphygmograph of v. Frey (made by Petzold of Leipzig) is preferred, but I am sorry to say that I have no means of forming a personal judgment of it.

What the sphygmograph really measures is the pressure of the pulse in the respective arteries. Therefore the instrument is only a refined [and recording] means of palpation. But it must be here emphasized that an absolute measure of the size of the pulse or of the internal pressure of the artery cannot be obtained in this way, as the height of the pulse-waves varies greatly with the position of the apparatus with reference to the artery and the position of the pad which receives the pulse. Hence it is well not to pay any attention at all to the height of the pulse-waves, but only to observe their form.

[1] In what way, see p. 219.

[2] See works upon Surgery.

In health the pulse-curve obtained with the sphygmograph shows elevations and depressions, ascending and descending lines, corresponding with the expansion and collapse of the artery. The expressions "apex-curve" (*cg*) and "curve at the base" (*b*) do not need further explanation. At both these points the curve stops only a very small portion of time.

The ascension line (*al*) is almost perpendicular; that is, the rise follows very quickly. The descent (*a*) is more drawn out and shows several small waves, which generally (not always) may be distinguished as a marked elevation (*r*), the backward-stroke elevation, caused by a wave of blood which results from the closure of the semilunar valve, and two (sometimes also three), or only one weaker, elevation produced by elasticity (*e*); the elastic secondary oscillation of the wall of the artery (according to Landois, but otherwise explained by others).

The elevation (*r*), the "recoil," has hitherto been regarded as a positive centrifugal wave due to the closure of the aortic valves. Recently v. Frey and Krehl have come to the conclusion that this explanation is not tenable—that the question in the "recoil elevation" is rather with reference to a centripetal wave which is reflected by the peripheral end of the circulation of the body, as by the closed end of a tube. The opinion formerly expressed that *r* was more marked the nearer we were to the heart would then probably have to be explained by saying that it was the summation of the waves which originate in various individual arterial regions, and are thence reflected.

But this view has met with strong opposition, and we too cannot avoid sharing in the opinions which have been brought against it, particularly by Hürthle, and are rather inclined to return to the old opinion.

However that may be, the quality of the "recoil elevation" has this diagnostic value: it increases with the diminution of the tension of the artery; its presence or absence, and in the former case its size, forms in itself a certain measure for judging of the blood-pressure; likewise, but in a reversed sense, when the "elasticity elevations" are very pronounced we must assume that there is considerable pressure. It is to



FIG. 70.—Normal pulse-curve in a healthy man, aged twenty-five years (after Eichhorst).

be remarked regarding the sphygmography of other arteries that *r* becomes more marked the nearer we go to the heart.

The following are the *essential pathological forms* of sphygmographic pulse-waves:

1. A descending line with several very marked elasticity elevations, but smaller backward-stroke elevations (often difficult to make out) which correspond with the increased tension in the aortic system (*lead colic, contracted kidney, acute nephritis*, etc.).

2. On the other hand, diminution of the elasticity elevation with more marked backward-stroke elevation shows diminished blood-pressure. Such increase of *r* is called "dicrotic," and the pulse "dicrotic pulse." Such a pulse, even if it is only moderately pro-

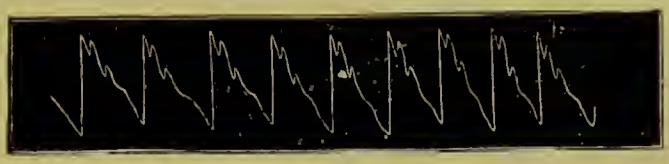


FIG. 71.—High-tension pulse.

nounced, can be recognized by *palpation*. It occurs in certain conditions which accompany a moderate diminution of strength of the heart, but especially a diminution of the tone of the arteries:

a. In *acute febrile diseases*, and indeed in so marked a degree and so early in *typhus abdominalis* [typhoid fever] that in diagnosis we may attach some, though not too great, value to this symptom.

b. In *chronic wasting diseases*, especially febrile—more than others in *tuberculosis*. Here, according to my observation, it is not infrequent.

c. In other weak conditions, as after great loss of blood, and in general in all forms of anemia.

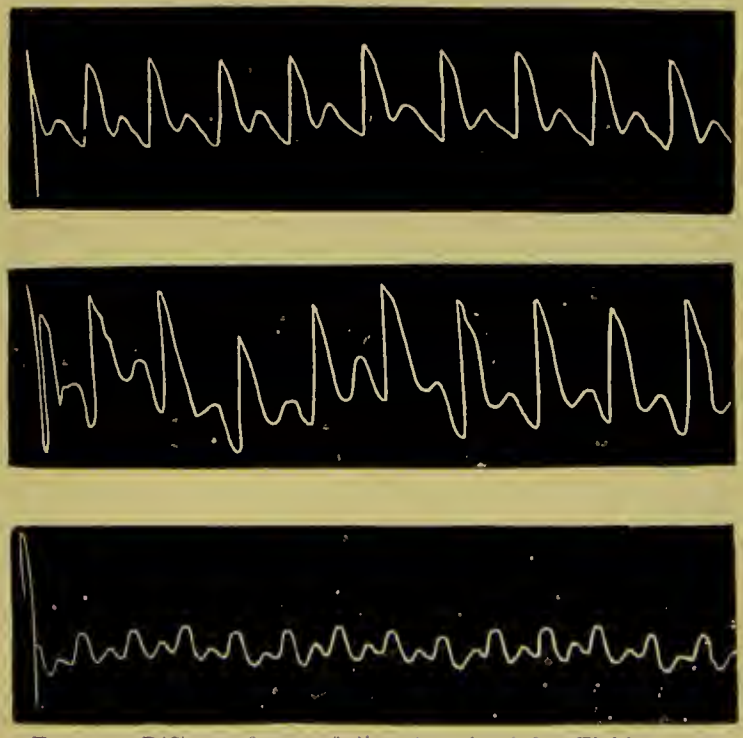


FIG. 72.—Different forms of dicrotic pulse (after Eichhorst).

The above curves show that in the dicrotic pulse the backward-stroke elevation may fall in the descending line (*subdicrotic pulse*), as well as in the middle of the basis curve (*complete dicrotic pulse*), likewise in the ascending line of the next following wave (*superdicrotic pulse*). The so-called *monocrotic pulse* (no visible backward-stroke elevation) is a sort of superdicrotic pulse.

What has been said in general regarding dicrotic pulse expresses the diagnostic value of all these forms of pulse.

3. To the *pulsus celer* corresponds a curve with a very steep ascending line and an unnaturally high apex-curve (in consequence of the quickness of the arterial diastole the recording lever of the apparatus is always thrown too high up). Moreover, the apex-curve is sharp-pointed, and the descending line is almost as steep as the ascending line. The elasticity elevations are marked. With pulsus celer due to *aortic insufficiency* there is, of course, no backward-stroke elevation, as the semilunar valve does not close.[1]

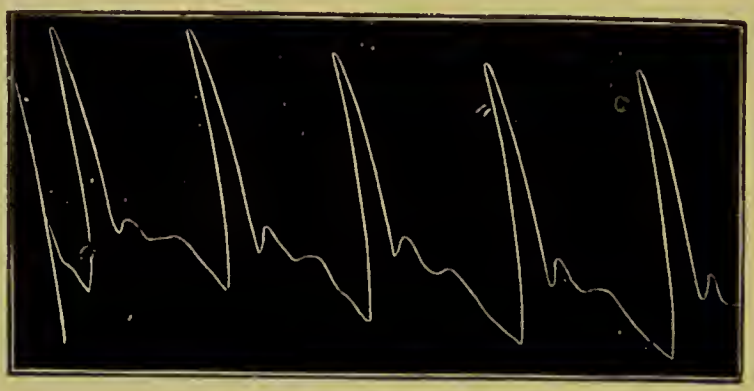


FIG. 73.—Pulse-curve in aortic insufficiency (after Strümpell).

4. *Pulsus tardus*, as in palpation[2] so in the curve, is the exact opposite of the preceding. With it there are usually more complete loss of the elasticity elevation and indistinct backward-stroke elevation.



FIG. 74.—Pulse-curve in stenosis of the aortic orifice (after Strümpell).

A peculiar combination of *pulsus celer* and *tardus* manifests itself with insufficiency and stenosis of the aorta.

In *pulsus tardus* the quickness of the apparatus is completely wanting on account of the slowness of the ascension; hence it always seems



FIG. 75.—Pulsus tardus in atheroma of the arteries (after Eichhorst).

small in comparison with the normal pulse-wave, and with that of pulsus celer[3] still smaller than is really the case.

It is quite impossible to form an estimate of *the size of the pulse*

[1] Compare what has been said on p. 209 upon Pulsus Celer.

[2] See p. 209.

[3] See this.

from the sphygmographic curve. The unequal pulse will generally be very beautifully delineated by the apparatus, but it cannot be more exactly depicted than it can be learned by exact palpation. It is true

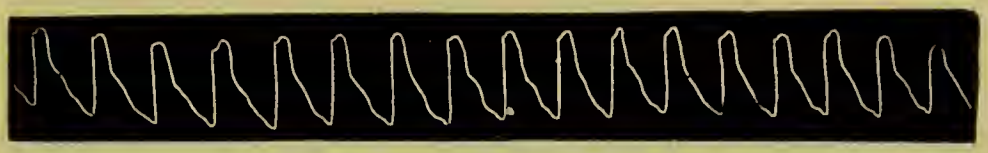


FIG. 76.—Pulse with anacrotic elevation in aortic insufficiency, with moderate stenosis of the orifice and arterial sclerosis.

that the apparatus includes small waves that the finger cannot recognize, but often these cannot be distinguished from the elevations indicating the backward stroke.



FIG. 77.—Pulse-curve with marked mitral stenosis (after Strümpell).

The *rhythm* of the pulse will, of course, even if only for a very short distance, be very well exhibited, and it is in this direction that the graphic delineation is very useful in giving instruction. But here sphygmography is wholly wanting for diagnostic purposes, since every notable useful irregularity can be felt just as well.

Annexed is an example of pulsus bigeminus (after Riegel).

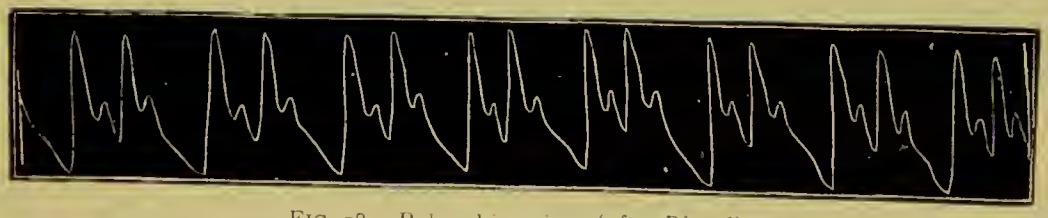


FIG. 78.—Pulsus bigeminus (after Riegel).

The application of the sphygmograph to both radial arteries simultaneously and comparison of the records will sometimes markedly increase the discrimination with reference to the symmetry of the radial pulses. Recently v. Ziemssen has shown that if the left subclavian artery is narrower at the point where it is given off from the aorta, the radial pulse of that side is very decidedly changed. He shows oblique lines of ascension, lowering, and retardation of the summit of the curve and monocrity, as is apparent from the accompanying figure.

Von Ziemssen designates this as *pulsus differens* in the narrower sense. This corresponds, as has been stated, with a narrowing of the subclavian at its origin, and it will be found in aneurysm of the arch of the aorta if associated with a stenosing endarteritis of the origin of the subclavian or a dragging of this vessel, or, finally, with compression of the commencement of the left subclavian artery. We may also expect to have the *pulsus differens* in compression of the left subclavian by any sort of tumor, by pneumothorax, and by a very large pleuritic

exudate. Aneurysmal dilatation of the aorta by itself—that is, without stenosis of the subclavian—does not seem to give rise to this pulse.[1]

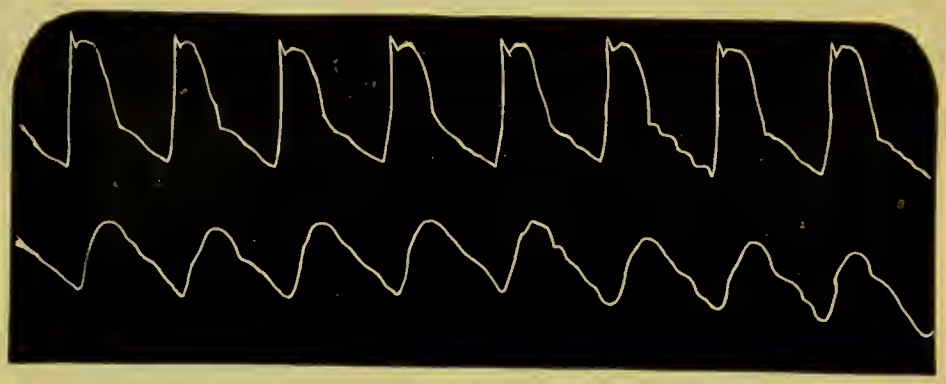


FIG. 79.—Pulsus differens: Aneurysm of aorta with stenosis of the mouth of left subclavian artery (after von Ziemssen).

Diagnostic Value of the Examination of the Pulse.

From what has been said it is sufficiently evident that for the purposes of diagnosis palpation of the radial pulse is preferable to sphygmography. The latter is more circumstantial, and gives, with a few exceptions, to one sufficiently practised in palpation no better result than the former. It very easily even deceives, especially regarding the size of the pulse, but sometimes also its form, from reasons that lie in the apparatus. Except in individual cases—as, for instance, when this question is in regard to *pulsus differens*—the great value of the sphygmograph for the clinician consists chiefly in its usefulness in giving instruction, for exhibiting a characteristic anomaly of the pulse to a large number of hearers, or it may serve to show a pupil what he ought to feel.

In what follows will be briefly indicated in which direction the examination of the pulse is of value for diagnosis, and how it can be turned to account:

1. Very often the pulse directly serves to determine the diagnosis—not that it alone is sufficient, but in connection with other phenomena it is. We are to bear in mind here what has previously been said regarding the behavior of the pulse in the various febrile diseases. But in diseases of the heart it especially has such an important place that a diagnosis is never to be made without taking into consideration the condition of the pulse.

In what follows is brought together what can be said regarding the behavior of the pulse in the most important of the diseases of the heart.

Mitral insufficiency: The pulse does not markedly or notably vary from the normal. But in addition the signs of hypertrophy of the right and left ventricles are present—systolic murmur at the apex.

Mitral stenosis: In classical cases, even with good compensation, pulse is small, unequal, or irregular, its frequency often much increased. Indeed, not infrequently these pulse signs are absent, and the pulse does not to any great extent depart from the normal. (In addition,

[1] Compare further p. 211; also p. 218 *f*, Aneurysm of Aorta.

there are signs of hypertrophy of the right ventricle and a presystolic murmur at the apex.)

Aortic insufficiency: Pulse is quick, frequency either normal or increased; generally equal and regular. In addition there are the signs of hypertrophy of the left ventricle and a diastolic blowing murmur at the aorta. (For the conditions at certain arteries, etc., see below, page 220 *f.*)

Stenosis of the aorta: Pulse is small, slow, normal or diminished in frequency, equal and regular. In addition, there are signs of hypertrophy of the left ventricle; only the apex-beat is often very strong and a systolic murmur heard over the aorta.

Myocarditis: Pulse is more or less small and soft, almost always irregular in quality, and generally so in time (here especially we have sometimes *pulsus incidens*, *bigeminus*). Frequency is increased, normal, or diminished. Nothing abnormal at the heart, or signs of dilatation of one or both ventricles; no murmurs.

Pericarditis exudativa: Pulse is strong if the heart remains so, generally somewhat quickened. In addition, at the heart all signs of its activity are diminished or removed by being covered over, marked dulness; in paralysis of the heart no pulse or very much quickened; sometimes *pulsus paradoxus*.

We are particularly to notice the opposite condition of the pulse in aortic insufficiency and stenosis, and also that in myocarditis the pulse may be the only sign.

In *combined valvular disease* the pulse is of importance in two ways: it betrays the existence of a second valvular disease besides the one already made out, as is especially the case in mitral insufficiency and stenosis. The latter near the former may be overlooked because very slight, or may even be entirely wanting, and because it produces hypertrophy of the right ventricle, which is also produced by the former, for there may be a very small, unequal, irregular pulse, which alone indicates the stenosis. Also, an aortic stenosis, besides insufficiency of the aorta, is sometimes certainly discovered only by the pulse, since there may be a weak systolic murmur at the aorta without stenosis. Thus the decision as to which cardiac orifice is concerned in the murmur, or whether we have one murmur widely conducted or two murmurs independent of each other, may be determined by the pulse.

Moreover, in a patient with combined valvular disease the pulse may very greatly assist in determining which disease is to be regarded as the more marked or important. This is especially true in insufficiency and stenosis of the aorta (the distinctness of the murmurs is, of course, not at all indicative;[1] also of the mitral or for combined disease of the aortic and mitral valves.

Thus we would diagnosticate a preponderating insufficiency and a very slight stenosis of the aorta when we have the signs of hypertrophy of the left ventricle, a loud sawing systolic and a very slight diastolic aortic murmur, and a pronounced *pulsus celer*. Thus, with the signs of aortic insufficiency and mitral stenosis a very small pulse points to the preponderance of the latter.

It is very difficult or even impossible to make a diagnosis of the

[1] See above.

particular heart-lesion, either from the general symptoms or from the pulse, so long as there is continued evidence of incompensation.

Moreover, in the cases where the heart and its action are concealed, especially in pericarditis exudativa, also in emphysema, sometimes in marked deformity of the thorax, displacement of the heart, tumors of the chest-wall, the pulse is the only sure sign of what work the left ventricle is doing. In pericarditis the contrariety that exists between a diminishing apex-beat, the slight, almost imperceptible heart-sound, and a strong pulse is sometimes a very important diagnostic point.

2. The pulse enables us to judge of the strength of the heart in all other possible—especially febrile—diseases. Even the first examination of the pulse furnishes, in this case, important information; but the signification of indications furnished by repeated examinations of the pulse (palpation and representation of its varying frequency upon the temperature-chart) becomes very much more valuable. These indications furnish still more important diagnostic points, some of which have already been spoken of. They have reference to the beginning of complications in acute infectious diseases, especially those affecting the heart, the lungs (which are very frequent), the kidneys (as after scarlet fever, when the pulse has greater tension and diminished frequency), and to the brain (decline in frequency in meningitis). Also, the effect of treatment, as of cold baths, may be determined partly by the behavior of the pulse; in general, it often determines the treatment.

We must also mention all diseases which in any way affect the heart, as pleuritis, pericarditis, peritonitis, in which the pulse, especially as a measure of treatment, has any part.

II. Other Phenomena in Arteries.

The Aorta.—Sometimes a pulsation is to be seen and felt in the neck: exceptionally also in health in consequence of higher location of the arch of the aorta; likewise in *hypertrophy of the left ventricle*, most marked in *aortic insufficiency*, since this causes a broadening of the commencement of the aorta; and, finally, in *aneurysm of the arch of the aorta.*

The occurrence of pulsation that can be seen and felt in the right second intercostal space is always pathological. It occurs in *hypertrophy of the left ventricle*, and also especially in *insufficiency of the aorta;* further, in *aneurysm of the aorta.* In rare cases, when there is marked hypertrophy, the second aortic sound may be felt, which, of course, can never be the case in aortic insufficiency.

In rare cases of aortic insufficiency the commencement of the aorta is accessible for percussion. It is to be remembered that here it is very much broadened, and to the right of the sternum, from the lower border of the second rib to the third rib, there is a small area of dulness. Sometimes over the aorta (in the right second intercostal space) in marked atheroma there ought to be heard a systolic murmur, even when there is no endocarditis aortica.

Aneurysm of the aorta requires a special description. It most frequently occurs in the ascending portion or the arch of the aorta, and

gives rise to the following phenomena: Only when the aneurysm is large is a swelling to be seen, and this, if present, is seen either above the sternum or close to the right of it. It generally pulsates—that is, becomes larger in all directions—with the systole of the heart. From stagnation[1] the enlarged veins of the skin are very early visible; later they may become red from inflammation or even be necrotic. In large aneurysm, under some circumstances, *when we palpate* we feel the pulsation, and besides, not infrequently, a peculiar whizzing or thrill. With large tumors, also, it further shows that the bones and cartilages over them have been absorbed. *Repeated measurement of the thorax* shows a gradual increase of the sterno-vertebral diameter. *Percussion* generally very early exhibits dulness, usually on the right, close to the sternum and over the manubrium; more rarely to the left of the sternum, and this either in connection with the area of heart-dulness or distinct from it. *Auscultation* not infrequently reveals the systolic whizzing, which has already been referred to as being felt, or also only two dull, impure sounds, or they may not be heard at all. The radial *pulse*, also the carotid, is not infrequently at an early stage upon one side smaller and a little later than on the other, in consequence of the compression of the particular branches of the aorta or distortion of their openings at the point of origin. Aneurysm of the ascending aorta affects the vessels of the right side, and aneurysm of the arch of the aorta sometimes affects those of the left side.[2] Not infrequently also there exists *insufficiency of the aorta* with hypertrophy of the heart. By all tumors in this neighborhood the heart may be crowded toward the left side.[3]

Aneurysm of the innominate produces about the same local symptoms as aneurysm of the ascending aorta, only generally somewhat higher up.

Aneurysm of the descending aorta (rare) may produce corresponding phenomena upon the left side, posteriorly, near the spine. The pulse in the abdominal aorta and its branches is usually later.

Aneurysm of the abdominal aorta (likewise rare) is generally at the level of the tripus cœliacus. It may be felt as a pulsating tumor in the upper part of the abdomen, and sometimes exhibits the whizzing mentioned above.

Considerable *stenosis, or even closure, of the aorta* at the junction of the ductus arteriosus is a very rare congenital condition, which is recognized by the fact that certain arteries furnish collateral circulation between the ascending aorta and the region of the descending thoracic aorta or the abdominal aorta. These collateral vessels become very much enlarged, and pulsate so as to be seen and felt. Diagnostically, the most important are the internal mammary, the anterior superior and inferior epigastric anteriorly, the transversus scapulæ and dorsalis posteriorly.

The Pulmonary Artery.—In very rare cases aneurysm of the pulmonary artery may give rise to almost the same symptoms as aneurysm of the aorta, except in being at the left of the sternum; that

[1] See p. 224.

[2] Compare *Pulsus differens*, p. 215.

[3] See further, under Examination of the Larynx, regarding the evidences of pressure by these tumors upon the trachea, upon the œsophagus, upon the left recurrent nerve (seldom the right). Regarding pressure upon the large veins, see p. 224.

is, if it is a question whether there is aneurysm of the pulmonary artery. A systolic murmur over the pulmonary artery may, besides, be caused by stenosis of the pulmonary opening or by *narrowing of the artery* itself. This may be congenital or be developed later, in the latter case by shrinking of the upper portion of the left lung. In such cases the second pulmonary sound is generally accentuated (hypertrophy of the right ventricle), and under some circumstances may even be felt.[1]

The Other Arteries.—Inspection.—Excepting during excitement of the heart (by mental excitement or physical exertion) we observe *in health* a visible pulsation of the carotid in the neck just under the angle of the jaw; also of the temporal artery. A marked pulsation of the carotid—especially when there is perfect mental and physical quietude, or, again, a general visible pulsation of smaller vessels, as of the temporal, the brachial, in the sulcus of the brachial muscle or at the bend of the elbow, of the radial, peroneal, dorsalis pedis—points to *hypertrophy of the left ventricle.* These abnormal pulsations are most marked in *insufficiency of the aortic valves* and in *arterial sclerosis;* in the first case on account of the fulness of the pulse, in the latter case on account of the thickened and stiffened vessels being prominent. In both classes of cases the smaller arteries are very *tortuous.*

Here also a *capillary pulse* is to be mentioned: alternating between marked fulness and emptiness of the capillaries, occasioned by the pulse in the arteries, the pulse may become visible under the finger-nails, more rarely over the tendons, in case these variations are connected with a large and quick pulse in the arteries, which, in turn, have large and quick alternations of size. Then, in examining the finger-nail, we see the red part rhythmically become alternately white and red—*capillary pulse of the bed of the nail.*[2] This is a sign of aortic insufficiency with marked hypertrophy of the left ventricle (which would also be present in some cases of marasmus).

Palpation.—Medium-sized and small arteries sometimes feel thickened and moderately stiff, or scattered in their walls we feel separate rigid patches, very like the plates of cartilage of the bronchial tubes or the rings of a small trachea ("goose's throat"). The latter become especially plain if we slip the tip of the finger up and down along the course of the artery. This is the condition in *arterial sclerosis.* Hence the vessels are often tortuous[3] and show variations of the pulse.[4] It is very easy to recognize arterial sclerosis in the temporal, radial, and brachial arteries. From the condition of these we can correctly estimate the condition of other arteries of the same size.

Palpation of the radial artery has already been described. Of the other arteries of the extremities the pulse of which we can feel in health, we may mention the brachial, in many persons the ulnar, the crural, the popliteal, and in most people the peroneal. Increased pulsation in arteries that can be felt, its occurrence in small arteries

[1] See above.

[2] [This is often an unfavorable situation for making the observation. Quincke, who first described the capillary pulse, now recommends rubbing gently a spot upon the forehead. —*Berliner klin. Wochenschr.*, Mar. 24, 1890.]

[3] See above.

[4] See this.

that can be felt, which in health are never made out, takes place in *aortic insufficiency*. A pulsation that can be felt in the dorsalis pedis artery is here very frequent, but the same thing may take place in still smaller arteries—in the digital, in the coronariæ labii inferior., superior., and the like. Very exceptionally in aortic insufficiency we may even observe an arterial liver-pulse; that is, a continuous to-and-fro swelling of the liver from the marked pulse in the arteries of the liver (quite like the venous liver-pulse).[1] Still more rare is an arterial pulse at the spleen.[2]

When in *symmetrical vessels*, like the two radials, we find a *pulse* that is *unequal* as to strength or time, we may generally conclude that there is a mechanical hindrance to the passage of the blood-current. We then have to seek toward the center from the weaker or later pulsating artery for a compressing tumor, thrombosis (autochthonous or embolic), or for an aneurysm. Moreover, there are observed variations of the pulse in symmetrical vessels, caused by vaso-motor influences from the nerve-centers. Finally, we must not overlook the possibility of anatomical variations.

Auscultation.—Mode of procedure: Here, it is to be understood throughout, the stethoscope is to be employed, and that ordinarily it is to rest upon the surface without pressure. We auscultate the carotid with the neck somewhat extended, but not stretched, in the intersterno-cleido-mastoid fossa or at the angle of the jaw; the subclavian, in the angle between the clavicle and the clavicular head of the sterno-cleido-mastoid muscle; the brachial, on the inner border of the biceps in the bend of the elbow, with the arm slightly extended; the crural, close below Poupart's ligament.

Normal Condition.—In health we usually hear over the carotid, as well as the subclavian, two sounds—one corresponding to the pulse, with the systole of the heart (the conducted aortic first sound and local diastolic sound in the vessel). In individual cases the first sound is impure or is entirely wanting. In health the diastolic heart-sound is never wanting. We sometimes hear over the abdominal aorta and the crural artery a sound which corresponds with the pulse, or, at any rate, arises locally from the tension of the vessels. We usually hear nothing over any of the small vessels. If we press with the stethoscope over the given vessel, then we hear the so-called acoustic pressure-sound, not alone over the aorta and subclavian, but also regularly over the abdominal aorta and crural artery, and usually, also, over the brachial. Thus, by moderate pressure over these vessels we hear a pressure-murmur corresponding to the arterial pulse; by stronger pressure, which almost, but not quite, closes the artery, this murmur is changed into a tone—pressure-tone. That these acoustic phenomena, resulting from pressure, are everywhere present are the chief reasons why the pathological conditions over the large vessels which are to be mentioned later have only conditional diagnostic value.

We must also mention a phenomenon frequently present in *healthy* children, called "cerebral blowing;" it is heard between the third month and the sixth year with the systole of the heart, or, more exactly, as a blowing corresponding with the carotid pulse, which is

[1] See p. 228.

[2] See under Examination of the Spleen.

heard sometimes light, sometimes tolerably loud, over the fontanelle while still open, but also sometimes after it has closed, and elsewhere over the head. Jurasz has in most cases found at the same time a blowing over the carotid, and thinks that the cerebral blowing is merely this murmur conducted upward. He explains the latter by the compression which the carotid sustains in the carotid canal during the development of the skull.

Pathological Conditions.—In *aortic stenosis* there will be heard over the carotid, in place of the first sound, a rough *systolic heart-murmur* (the stethoscope must rest very lightly).

In *aortic insufficiency* the second sound of the carotid and subclavian is wanting, or it is replaced by blowing with the diastole of the heart (rare). This, as well as the systolic murmur previously mentioned, is conducted from the mouth of the aorta. The former, arising in a current of blood flowing forward, would naturally, as a rule, be more loudly conducted than the latter, which comes from a backward-flowing blood-current.

Sounds in such arteries as in health very seldom or never furnish a sound accompany *aortic insufficiency*, being produced by the quick and strong tension of the vessels during their diastole. We then hear a sound corresponding with the pulse over the crural, brachial, radial, even the ulnar, peroneal, dorsalis pedis arteries; sometimes even over still smaller vessels. A sound is also observed over the crural in *high fever*, as well as in *anemia* and *chlorosis* (and as well in some healthy persons).

A double sound over the crural artery (Traube) is heard in individual cases of aortic insufficiency. But this phenomenon has also, although very exceptionally, been observed with mitral stenosis (Weil), likewise in lead-poisoning (Matterstock); lastly, in pregnancy (Gerhardt). Much more important is the double murmur which is heard when considerable pressure is made with the stethoscope—*Duroziez's double murmur*. In the experience of observers, thus far, this occurs only with aortic insufficiency, and this when there is good compensation; and this has all the greater significance from the fact that it is decidedly more frequent than was previously supposed.

Double sound, as well as *double murmur*, can only occur when there is a large and quick pulse. In the first phenomenon the double sound is caused by the sudden collapse of the artery; with double murmur the second murmur is probably to be explained by the short reflux blood-current which may be assumed to flow into the large vessels when there is aortic insufficiency (?). A double sound can also be heard over the crural artery if one of the two sounds, or even if both sounds, arise from the crural vein.[1]

A *systolic subclavian murmur* is sometimes heard on both sides, or sometimes only on one side (especially the left), as a very disturbing addition to the breath-sounds at the apex of the lungs. It is stronger, or perhaps only to be heard, toward the end of inspiration. When it occurs upon both sides, as a rule, it does not indicate a pathological condition; when unilateral, it also has no significance, and yet it always gives the suspicion of phthisis, with which we often meet it. It is ex-

[1] Regarding this, see next chapter.

plained by a temporary pulling or bending, and hence narrowing of the subclavian artery during deep breathing. In phthisis this is caused by adhesion of the pleural surfaces at the anterior surface of the apex of the lungs. We do not know exactly why this murmur occurs also with persons apparently perfectly healthy, but it may possibly be from the same cause.

Loud *blowing murmurs* over the thyroid glands sometimes occur in all forms of *struma*. These murmurs may be felt. They are not infrequent with struma of Basedow's disease, but here they are caused by the excited action of the heart.

The murmurs which in some cases are heard over aneurysm have been already mentioned.

EXAMINATION OF THE VEINS.

We examine chiefly, in many cases exclusively, the jugular veins (external and internal in the neck), but also the cutaneous veins of the body and extremities. Only in special cases (thrombosis) do the deep veins of the extremities become accessible for examination. The ophthalmoscopic examination of the ophthalmic veins does not come within the scope of this book. It is important that we are able to judge of the abnormal fulness (engorgement) of certain deep veins by its effect upon particular internal organs, as enlargement of the liver and spleen, also ascites, and, lastly, the suppression of urine.[1]

The examination of the veins is made by inspection, or sometimes by palpation and auscultation.

Inspection and Palpation of Veins.

By these means we ascertain the degree of fulness, the condition of the circulation, and, under some circumstances, the existence of venous thrombosis. An unusually empty condition of the veins does not come under consideration. This would also be very difficult to determine, for the reason that even in health, especially in fat people, the superficial veins may be indistinct or entirely invisible. It remains to describe—1. Increased fulness of veins; 2. Circulation in the veins of the neck; 3. Circulation in the other veins; 4. Venous thrombosis.

1. Increased Fulness of Veins.—This is the result of stoppage of the blood in its course toward the centre. It is general or local according to the cause of the engorgement, whether this be central or at some place in the course of the nerves that control the circulation.

General increased fulness is the result of general venous engorgement. We first recognize it by the swelling of the internal and external jugular veins upon both sides. The first of these is usually visible in health (but not always, especially in fat people), coursing obliquely over the sterno-cleido-mastoid muscle. When the head is turned toward the opposite side it usually swells still more. With the increased fulness it becomes distinct, perhaps can be felt. With normal fulness the internal jugular cannot be made out, situated, as it is, under the sterno-cleido-mastoid muscle, where it is divided into the

[1] See under Enlargement of Liver, of Spleen, also Ascites and the so-called Urine of Engorgement.

clavicular and sternal portion, just in the angle between these, at the bottom of the intersterno-cleido-mastoid fossa. Where it passes into the bulbus jugularis it has a valve (ordinarily exactly at the upper border of the sterno-clavicular articulation, but sometimes, especially in consequence of the engorgement, located somewhat higher). Abnormal fulness of the jugular vein fills up the intersterno-cleido-mastoid fossa or it may cause a projection there. Dorsal posture increases the fulness. Fulness of the cutaneous veins of the trunk and extremities, not occurring without general engorgement, is usually not so pronounced as that of the veins of the neck, especially on account of the marked edema which accompanies the congestion. Important associated symptoms of general engorgement are *cyanosis*, *edema*, *effusion into the cavities of the body*, *enlargement of liver and spleen*, *disturbance of the bowels*, *and so-called urine of engorgement*.[1]

This condition arises when the right heart is not able to propel the required quantity of blood into the lungs. It occurs in various *diseases of the heart*, in *emphysema of the lungs*, and in all the conditions that lead to marked *interference with the action of the heart*, especially pericarditis. The most marked engorgement occurs in general when the right side of the heart is paralyzed after it has been obliged for a long time previously to meet unusual demands, and hence has become hypertrophied; hence with *mitral* and, more rarely, *pulmonary defects* and *emphysema*, and likewise in the very rare *tricuspid stenosis* and *insufficiency*.[2]

General abnormal fulness of the veins may also be the result, exceptionally, of diminished flow of blood from the two cavæ into the right auricle in consequence of pressure by a mediastinal tumor.

Local increased fulness of the veins may be caused by a considerable narrowing or closure anywhere of a venous trunk by a thrombus or by compression. The larger the vessel thus affected, the more extensive the area of abnormal fulness. Thus, sometimes abnormal fulness of the jugular and its branches, also of the ophthalmic vein (recognized by the ophthalmoscope), will be caused by a mediastinal tumor which presses upon the cava. Also the superficial veins of the skull between the ear and the fontanelle will become distended and tortuous if the longitudinal sinus of the dura is stopped. Fulness of the veins of an arm points to compression of the axillary vein (generally tumors or scars from operations in the axilla). The swelling of the veins of the skin over or on either side of the sternum is a very important early sign of mediastinal tumor. The cutaneous veins of the leg are enlarged when there is thrombosis or compression of the femoral vein of that side. The veins of both legs may swell as the result of double thrombosis or compression of the vena cava inferior or both iliac veins (ascites, tumors). In all these cases there may be local edema.[3] This may even give a better and earlier sign of local engorgement, but, on the other hand, it may conceal the fulness of the veins.

In the majority of such cases the cutaneous veins supply the necessary collateral circulation. But this is especially the case in engorgement of the portal vein,[4] whether due to cirrhosis of the liver or com-

[1] See this. [2] See under 3, p. 229. [3] See this.

[4] See also Enlargement of the Spleen, and Ascites.

pression or thrombosis of the portal trunk. Here we may see the abdominal veins enlarged, part of which go upward to the thorax and part downward to the inguinal region. In individual cases there is a crown of such veins around the navel—"*caput Medusæ*"—since the umbilical vein, remaining open, receives a part of the overflow of blood which the portal is not able to carry.

The fine dendritic vein-nets which are frequently seen on the lower portion of the chest, and here and there also on the back along the course of the lower boundary of the lungs, more rarely along the sternum or in the fossæ supraspinatæ, are difficult to interpret. They appear most frequently in emphysema of the lungs, and also in adhesive pleuritis. I have lately found them three times within a short period in cases of adhesive pericarditis on the boundary between the heart and lungs. Probably they are always signs of collateral circulation, which in pleural coalescence it is certainly not difficult to understand, but more difficult in emphysema. However, such vein-nets are sometimes also seen in persons in whom no anomaly of the thoracic organs can be found.

Very extensive enlargement and tortuosity of a large part of the cutaneous veins of the trunk or of the chest (generally symmetrical), or enlargement of single cutaneous veins of an extremity, also occur without any possible assignable cause (perhaps closure of a deep branch), so that, according to the views of the present day, it is to be regarded as an independent primary alteration of the respective veins. Whether this alteration is to be considered as a congenital disposition or an anomally gradually acquired later, possibly a kind of chronic phlebitis, cannot yet be decided.

2. Phenomena of Circulation in the Jugular Veins.—Respiratory Motions.—The suction action of the chest with inspiration causes a rapid emptying of the blood from the veins of the body into the heart during inspiration, as well as during expiration. On the other hand, a forced expiration, likewise strong effort, and very especially the increased internal pressure within the chest which takes place in coughing before each cough-impulse, check the discharge. The alteration in the fulness of the veins in the neighborhood of the heart which is thus caused is usually only to be observed in the jugular veins. But in normal fulness of these veins the simple respiratory oscillation of their volume is not noticeable. Such veins only distinctly swell with marked pressing and coughing (whooping-cough), and then the veins of the face become very full. Yet when the veins of the neck are constantly abnormally full or engorged, then in ordinary breathing they show a corresponding to-and-fro swelling, and with forced expiration, pressing, or coughing they stand out very distinctly. The *bulbus jugularis* may then appear as a round bunch between the heads of the two sterno-cleido-mastoidei muscles; but even the whole internal jugular may swell and contract if the valve over the bulb does not close. This phenomenon occurs in the most marked degree with the labored expiration of emphysema. Here, also, in very rare cases, this variation in the fulness extends to the cutaneous veins of the face, the chest, and arms.

The opposite condition of the veins of the neck, becoming tumid

with inspiration and emptying with expiration, may be caused by fibroid mediastinitis (mediastino-pericarditis). The cause of the phenomenon, like that of *pulsus paradoxus*,[1] is the traction and bending of the large vessels during inspiration (Kussmaul).

Venous Pulse.—Circulatory movements in the veins of the neck, which directly or indirectly depend upon the action of the heart, and hence are rhythmic, are designated as venous pulse. This motion may be communicated to or really be in the vessels (autochthonous, real pulse). The former is only the pulsation in the carotid communicated to the internal jugular, which shows most frequently and plainly when the carotid pulsates very strongly or when the internal jugular is very full, or if both conditions exist.[2]

We divide the real venous pulse, pulsation in the veins of the neck, into that which occurs in health, the so-called "normal" or negative, and the positive, which is always pathological. The normal venous pulse is presystolic, and usually is only observed in the external jugular. It would be best designated as a collapse of the vein accompanying the systole of the heart; for the external jugular, in exact correspondence with the apex-beat and the carotid pulse, quickly empties itself, and

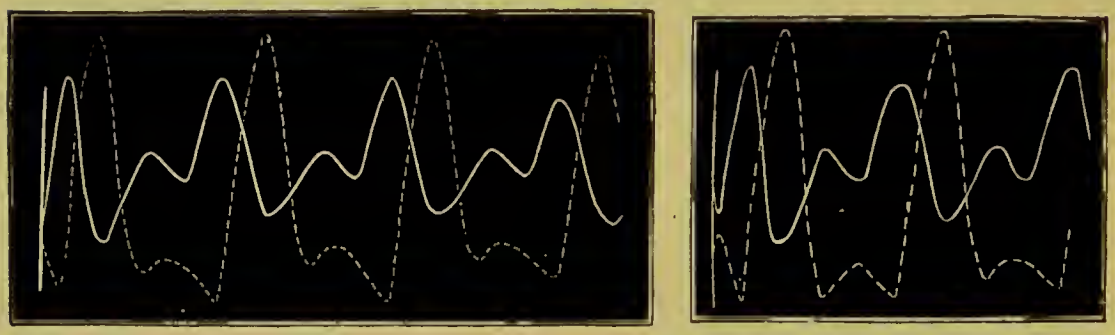


FIG. 80.—Normal venous pulse or venous collapse with systole of the heart, and (broken line) carotid pulse (after Riegel).

immediately again slowly fills, sometimes visibly in two intervals, so that it attains its complete distention before the next systole of the heart, and hence is presystolic.

This phenomenon depends upon the part the auricle plays in the action of the heart: during the ventricular systole it is in diastole, and thus favors the flow of blood from the veins. Shortly after the beginning of the ventricular diastole it begins to contract, and thus the flow of the venous blood from the cava into the auricle is impeded. It seems to me that the first elevation of the ascending side of the tracing of the curve of the venous pulse has not yet been explained. In health this pulse is seen to a very small, scarcely noticeable, degree; it is beautifully seen in dogs when the jugular is laid bare. In healthy persons, without any known reason, it is in some cases strong enough to be observed. But sometimes it is still stronger when the external jugular is abnormally full, hence in engorgement. Often this pulse occurs only indistinctly; its rhythm is difficult to recognize, and it is also affected by the pulsations of the carotid. Then we speak of *undulation in the veins of the neck.*

[1] See this. [2] For distinction between this and genuine systolic venous pulse, see p. 227.

The *positive venous pulse is systolic*, hence is contemporaneous with the carotid pulse. It is a pathognomonic sign of insufficiency of the tricuspid valve, and is caused by the contraction of the right ventricle, which causes a regurgitant positive blood-wave into the cava and its nearest branches through the imperfectly closed right *ostium venosum*. It first and most markedly appears in the internal jugulars or their bulb, and generally only here. The very direct course of the innominate and right jugular from the cava causes the right jugular vein to show the phenomenon more frequently and stronger than the left.

If the valve of the vein closes above the bulb of the jugular, then the regurgitant wave ends there. This pushes the bulb up and distends

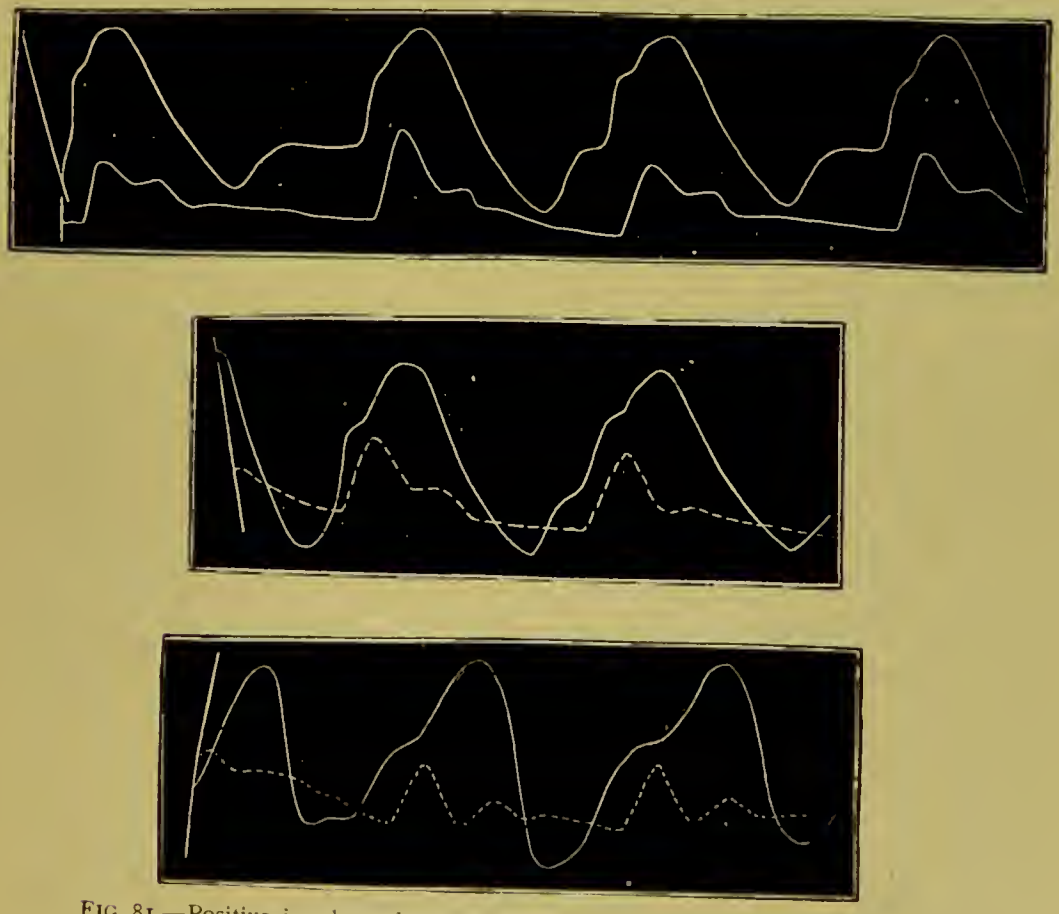


FIG. 81.—Positive jugular pulse compared with (*C*) carotid pulse (after Riegel).

it, and it is then seen, enlarged and pulsating, in the intersterno-cleido-mastoid fossa (*bulbar pulse*). The bound of the pulse-wave against the valve sometimes causes a *valvular sound in the jugular*. But ordinarily the valve is insufficient from previous engorgement (or is congenitally so), or it becomes so from the distending action of the pulse, and then the pulse-wave passes into the internal jugular, and exceptionally also into its branches in the face. This systolic pulse must likewise be supposed to be propagated to a certain extent also in all other veins that are directly given off from the cava; but they cannot be examined except in a large venous territory—the veins of

the liver. Here the pulse manifests itself by a constant systolic swelling and diastolic collapse of the organ—*the venous liver-pulse.* Palpation of a liver thus constantly enlarged frequently shows the phenomenon of systolic venous pulse to a high degree.

The systolic jugular pulse may be graphically represented, as is shown in Fig. 81.

The mode of procedure in palpating the liver-pulse is as follows: One hand is placed upon the right hypochondrium or the epigastrium; the other is passed around the chest at the level of the eleventh and twelfth ribs posteriorly. We can then feel that the organ is systolically enlarged, and thus we may avoid confounding it with lifting up of the liver by the aorta or even with marked epigastric pulsation. Moreover, we recognize the liver-pulse in this way easier—that is, sooner—than by simply palpating in front. The liver is usually enlarged, almost always by the previously existing engorgement;[1] at least, it immediately becomes so if tricuspid insufficiency occurs, as we very distinctly observed in a case of mitral insufficiency and stenosis, in which relative tricuspid insufficiency occurred, then subsided, and again reappeared.

Arterial liver-pulse is exactly like venous liver-pulse in its phenomena (in aortic insufficiency).[2]

For the production of a recognizable venous liver-pulse, as well as a strong jugular pulse, there is, of course, required a certain moderate (and, if it has not been met with before, also it must not be too frequent) action of the heart. As the heart grows more and more weak the liver-pulse fails, and the jugular pulse gradually becomes smaller and more slow, until finally there is only a slight to-and-fro movement of the vein.

In order to make a *differential diagnosis of the different kinds of pulse in the veins of the neck* it is necessary to bear in mind the following: 1. The transmitted pulse will be best distinguished from the positive real pulsation occurring at the same time with it by placing the finger or, better still, a plexímeter, with its edge in the middle of the neck, upon the vein: if the pulsation is communicated, it disappears in the central empty portion and becomes more distinct in the periphery from the engorgement of the distended portion; on the other hand, a positive genuine pulse remains centrally unchanged. 2. The negative true pulse is distinguished from the positive and from the communicated pulsation generally by comparison with the apex-beat, as well as by comparison with the carotid pulse. (We seize the left carotid and at the same time observe the right jugular.) It is also to be observed that with the negative pulse the collapse of the vein is usually quick, and that it refills slowly. In this way, with a little practice, one can often immediately judge correctly.

In order more exactly to observe and study these phenomena it is well to have the patient for a time breathe very superficially, or, if possible, to hold the breath, so as to eliminate the respiratory to-and-fro swelling of the veins.

We must still mention some occurrences that are extremely rare or are of very little diagnostic value:

[1] See Enlargement of the Liver.

[2] See p. 221.

Diastolic collapse of the cervical veins (Friedreich), which looks very like systolic venous pulse, sometimes occurs in adhesive pericarditis and fibroid mediastinitis, and is connected with systolic drawing-in in the neighborhood of the heart, which occurs with this condition.[1] The springing forward of the heart in the diastole, together with the forward movement of the anterior wall of the chest, probably produces an aspiration of the contents of the large veins.

Systolic venous pulse may exceptionally occur with mitral insufficiency and open foramen ovale: through the latter and the left ostium venosum the contraction of the left ventricle produces a recurrent pulse-wave in the cavæ and their nearest branches (very rare, being thus far only observed in one case).

Double positive venous pulse (Leyden) is observed in hemisystole.

3. Phenomena of Circulation in Other Veins.—Systolic true pulse may, as has already been mentioned, be propagated to the veins of the face, but this is rare. In individual cases it has even been observed in the cutaneous veins of the arm, in the small branches of the internal mammary (of which I have seen one case), in the vena cava inferior (Geigel), etc.

The so-called *progressive* or *ascending venous pulse* (Quincke, Holz) has been seen in the veins of the hand and the back of the foot and in those of the forearm up to the elbow. This phenomenon may be met with in very different conditions: it seems to appear principally when the vessels of the extremities have a diminished tonus, the veins more or less full, and when the heart acts vigorously. Quincke has observed the ascending venous pulse in febrile states of every kind, in cerebral and spinal diseases, in chlorosis and anemia, finally in healthy subjects during hot weather. Holz and Senator have seen it in pseudo-leukemia and leukemia.

Probably it can scarcely be explained otherwise than as an arterial pulse propagated through the capillaries; but opinions still differ about the real conditions of its occurrence, and also as to its prognostic significance. As is evident from what has been said, as yet it cannot be turned to account diagnostically.

4. Venous Thrombosis.—The transformation of the soft venous tubes into firm round cords that can be felt exhibits venous thrombosis. The thrombosed vein may often also be perceived by pressure. In internal medicine, of especial interest and importance is thrombosis of the large veins of the lower extremities as it sometimes occurs in the course of severe *acute infectious diseases* as the result of chronic invalidism, and in marasmus of the aged. Frequently, but never while resting in bed, it occurs in the edema of engorgement in the affected limb.

It is important to touch such veins very carefully in order not to push off a piece of the thrombus. A piece torn off from the central end of the thrombus may be carried to the right ventricle, and from thence produce an embolism of the pulmonary artery.

[1] See pp. 176, 177.

Auscultation of Veins.

1. Sounds and murmurs of short duration are sometimes heard over the jugular and crural veins.

In tricuspid insufficiency there is a systolic recurrent blood-wave, which, by its impulse against the closing valve above the *bulbus jugularis* and against those in the crural vein at Poupart's ligament, and also by the sudden tension of the vein itself, causes a sound which will be heard by very lightly placing the stethoscope at these points. But a sound has also been heard where the crural valve was defective. In such cases it must be alone caused by the sudden tension of the venous tube. If these valves are insufficient, there may be a corresponding short murmur (very rare).

The jugular sound generally accompanies the bulbar pulse of tricuspid insufficiency. A venous sound over the crural is, however, rare, because the recurrent wave only exceptionally reaches this vessel. Quite exceptionally with tricuspid insufficiency there may be a double sound over the crural vein, indicating first auricular, then ventricular, contraction (Friedreich). It can be distinguished with certainty from the sounds, double sounds, and murmurs of the crural artery only when there exist signs of aortic or tricuspid insufficiency (hence, how small is the diagnostic value of these phenomena!). Crural, arterial, and venous sounds may be combined when there exists at the same time aortic and tricuspid insufficiency.

Now and then, even in health, especially in thin persons, a sound is produced over the crural vein by sudden straining or coughing (expiratory valvular sound in the crural vein—Friedreich).

2. A continuous murmur, designated as *venous humming*, *venous murmur*, or *buzzing*, is often heard in anemic, and especially in chlorotic, patients, but sometimes also in many healthy persons, over the jugular veins. It is usually louder on the right side. It sounds like a regular humming or a very fine whizzing, or like the humming of a top. If it is very marked, it can also be felt. The murmur is caused by the whirl in the blood as it flows from the narrow jugular into its wider bulb. The whirls are the more marked the more rapid the stream, and hence the murmur becomes louder in deep inspiration; and for the same reason it is generally louder in the upright position than when lying down. And likewise it is not infrequently louder in the diastole than in the systole of the heart. Also, the predominance of the right jugular over the left is explained by the difference in the rapidity of the current caused by the different shape of opening into the cava.[1] This murmur will be increased by slight compression, as may be produced by the stethoscope or by turning the head to the opposite side. This latter effect comes from the tension of the *fascia colli*, and probably also from the contraction of the omo-hyoideus muscle.

As to what the occurrence of this murmur means, we must rest upon the old idea that it chiefly occurs with anemic, and especially chlorotic, patients. Friedreich's claim that it is more marked in these cases, while in health it is usually only to be heard as a soft humming, seems to me

[1] See above, p. 227.

to be very far fetched. Strictly speaking, no diagnostic importance is to be attached to this phenomenon.

A similar murmur occurs exceptionally in other veins, and it is to be noted almost exclusively in anemia; thus, in the large veins of the extremities and also in the intrathoracic trunks. Here the murmur is always much stronger during the heart's diastole, and can thus appear to be interrupted. It has already been mentioned that Sahli declared the anemic heart-murmurs to be in part propagated from the venous trunks in the chest.

EXAMINATION OF THE BLOOD.

Preliminary Remarks.—We can only approximately determine *the total amount of blood* in a healthy person. Its direct determination is of course impossible, and we are compelled to form approximate conclusions from animals. In mammals the quantity of the blood fluctuates considerably: it is between one-eleventh and one-twenty-third of the weight of the body. In dogs the variation is from one-eleventh to one-eighteenth of the weight. Of the quantity of the blood in diseased conditions we know from autopsies scarcely more than that it is diminished in a very conspicuous manner after severe hemorrhages and after the loss of large amounts of the water of the organism, as in cholera Asiatica and other severe diarrheas. But at the bedside we are still less able to estimate the quantity of the blood of the patient, even approximately. We certainly know that in genuine anemia from hemorrhage the color of the skin and of the mucous membranes becomes paler and the pulse smaller; but only under quite exceptional circumstances can we reason backward, *vice versâ*, from these signs to a diminution of the quantity of the blood, because paleness and weakness of the pulse may also be produced by disturbances of the circulation, and because paleness may be caused by a watery quality of the blood in itself—*i. e.* hydremia without anemia.

Thus we know almost nothing of the quantity of the blood of the patient, and the conception "anemia" has a very defective foundation. Apart from particular cases mentioned above, perhaps upon the whole it never exactly applies, because it has been proved experimentally that the blood in a high degree has the capability to balance a diminution of its quantity by quickly absorbing water. Nevertheless, if the expression "anemia" is used, it is only because it has become naturalized. According to our present views it corresponds with the conceptions of hydremia, hypalbuminosis, diminished hemoglobin on the one hand, and diminution of the red cells on the other. It must be pointed out that the last-named state need not go quite parallel with the first-named. In respect to the so-called anemias, it is therefore of interest for the diagnostician to know in the first place the percentage of water and albumin, and particularly the percentage of hemoglobin—*i. e.* coloring-matter of the blood—and the number of red and white blood-cells.

Besides, there are conditions in which *the spectroscopical behavior of the blood* is altered—conditions in which the form, size, and structure of the red and white cells are altered. There are also pathological admix-

tures of different sorts, and finally certain less important chemical alterations, as, for instance, decreased alkalescence, etc.

The examination of the blood must therefore include a number of points of view, but they do not all of them always come into consideration. Frequently we may be content with a very simple procedure, according to the result of which, and according to the remaining factors of the patient's condition, further investigations must be made.

Anticipating somewhat, we here give a synopsis of the different steps in making examinations of the blood:

1. The most simple procedure, which is often sufficient, and where it is not sufficient gives hints as to further examinations, is: determination of the percentage of hemoglobin (Gowers-Sahli's hemoglobinometer) and inspection of a fresh microscopical preparation.

2. A procedure which is sufficient for most pathological conditions of the blood: besides determining the amount of hemoglobin and the inspection of the fresh microscopical specimen, we are to count the red and white blood-cells and determine their proportion to each other. We are also to make and inspect an eosin-hemotoxylin preparation.

To these there follow in succession: (*a*) either exhibition of Ehrlich's granulations and of the nuclear structure of the leucocytes; (*b*) or, as may be necessary, other special methods, as, for instance, a study of the micro-organisms.

Sometimes from the beginning we have only to examine for micro-organisms (recurrens [spirillum of relapsing fever], anthrax, etc.); sometimes the attention is principally directed to the spectroscopic quality of the blood (certain cases of poisoning), etc. These details will become clear from what follows.

Regarding the value of centrifuging the blood by means of the hematocrit we have no basis for a personal opinion, though we doubt whether the method will have a lasting value for the diagnostician.

The methods of obtaining blood differ according to whether a smaller or larger quantity is desired. For most purposes it is sufficient to obtain the blood by a puncture in the tip of the finger or lobe of the ear. After having used it for many years, we can most strongly recommend the scarificator devised by Francke (made by Katsch in Munich), and we particularly emphasize these points in regard to it: it can be easily disinfected; it can be used for the smallest punctures; it can be so arranged that only the fine point of the lancet penetrates the skin. A particular advantage we found in the fact that, instead of using the finger-tip or lobe of the ear, we may use a place a very little larger, and may obtain the drop of blood at a place less rich in blood-vessels, as somewhere on the arm. This is in every respect a better place. If a larger quantity of blood is required, as is desirable in making cultures and is indispensable for quantitative chemical analysis, we recommend the very simple and perfectly safe method which v. Ziemssen has recommended, which consists in removing the blood by aspirating the median vein.[1]

1. **Color (Amount of Hemoglobin); Spectroscopic Character of the Blood; Density of the Blood.**—Blood taken directly from a healthy person is of a recognized color: if arterial, it is brighter, rich in

[1] See p. 251.

oxygen—that is, rich in oxyhemoglobin; if venous, it is darker, bluish-red—that is, it is poor in oxygen. The marked deficiency of oxygen in the blood of a person suffering from dyspnea or venous engorgement, or both, makes the blood very dark. In carbonic-acid poisoning the blood is bright cherry-red; from chlorate of potash, anilin; and in severe poisoning by hydrocyanic acid and nitrobenzol it is brownish-red or chocolate color. In severe anemia and chlorosis (hydremia) the blood is watery; in marked leukemia it looks a peculiar whitish-red, as if mixed with milk, or chocolate color.

These changes in the color of the blood all have an effect upon the color of the patient's skin, as has already partly been mentioned. Hence patients with carbonic-acid poisoning look strikingly rosy, while in poisoning with chlorate of potash, anilin, and nitrobenzol the skin and mucous membrane are a peculiar grayish-blue or black color. These discolorations of the skin, as well as the differences in the color of a drop of blood obtained by pricking with a needle, have too little distinction to be directly of diagnostic use. But, especially with regard to the poisons that have been mentioned, if they are recognized as unusual, they demand that a timely and thorough examination of the blood be made by the spectroscope or microscope. In this lies the great value of a knowledge of these discolorations.

For recognizing *hemoglobinemia* (from the hemoglobin that appears in solution in the serum of the blood originating from the red blood-corpuscles) it is necessary to employ a wet cupping-glass, or to take blood from a vein after the method of v. Ziemssen. The blood thus withdrawn is allowed to stand covered for twenty-four hours, if possible in an ice-chest, and then the serum, separated from the coagulum, is to be examined. That from normal blood is yellow, in hemoglobinemia it is rubine-red, and in the spectroscope gives the bands of oxyhemoglobin.[1]

Approximative Determination of the Amount of Hemoglobin.—A diminution in the amount of the hemoglobin may be conditioned upon a diminished number of red corpuscles or upon a decrease in the amount in single corpuscles, or upon both.[2] The color of the skin is a very unsafe index of the percentage of hemoglobin in the blood. The color of the mucosæ also is often misleading, but in any case is always a very inexact index, for reasons we have already pointed out. Therefore, lately reliance is more and more placed on the examination of the blood itself. In extreme anemia the drop of blood which exudes from a wound made by a needle-puncture on the finger appears distinctly pale to the practised eye, and enables it to recognize without doubt a diminished percentage of hemoglobin. To make possible, however, the judging by the eye of the percentage of hemoglobin in a drop of blood, even in slighter variations from the normal, technical aids are absolutely necessary. Of late there have been constructed to this end a number of instruments. We mention here only two, which we most strongly recommend; Fleischl's hemometer and Gowers's hemoglobinometer. Both of them possess by no means absolute exactness, but are sufficiently accurate for the purposes of practice, and are comparatively simple and quick in giving results. Formerly we always used only

[1] See below, p. 236.

[2] See below.

Fleischl's instrument, but lately, in consequence of the earnest recommendation of Sahli, we have frequently used also the much cheaper and still more simple instrument of Gowers. We recommend the latter instrument to every practitioner for widest use in diagnosis of anemia and determining its degree. The principle of the hemometer of Fleischl is as follows:

A certain very small quantity of blood (obtained by a prick of a fine lancet, or, better, by means of a [Francke] scarificator), is thinned by a definite quantity of water, and then by lamp or gaslight the color of this mixture is compared with the color of a glass wedge which has been colored with Cassius's gold purple and carries a movable scale. Upon this scale the figure 100 corresponds with the intensity of color of a mixture of normal blood. Material that has less intensity has the numbers 90, 80, etc., down to 10, thus giving directly the percentage relation of the mixture of blood that is being examined to that of normal blood with reference to the quantity of hemoglobin. Thus, 50 indicates, if the mixture of blood has been properly prepared and corresponds in color with the color of the glass wedge at that point of the scale, that this blood contains only 50 per cent. of the normal quantity of hemoglobin.

This instrument gives inexact results, varying in extent with different specimens, but, except when the amount of hemoglobin is greatly diminished, the inexactness is not sufficiently marked to vitiate the conclusions. Dehio gives the following table of variations with his instrument. (The findings were too small by the amounts represented by the figures in the second column.)

With 90 per cent. of hemoglobin	0.4
With 70 " "	2.8
With 50 " "	4.5
With 20 " "	5.5

The principle of Gowers's instrument is similar: it is to determine how much a certain very small quantity of blood must be diluted to have the mixture agree in intensity of color with a normal solution, which consists of picric acid and carmin in glycerin. This solution, however, can be used only for comparison in daylight. For use in artificial light Sahli has made another solution which he calls "normal solution to Gowers's hemoglobinometer." (It is sold by the optician Hotz in Berne.) Water is used as the diluent for the blood.

Method of Using Gowers's Hemoglobinometer.—A small wooden block forms a stand for the tubes *b* and *c* (see Fig. 82). The tube *b* is closed and contains the test liquid, picric-acid-carmin solution. The tube *c* is open at the top and serves for the reception of the blood-mixture which is to be examined. On this tube is a scale which gives the percentage of hemoglobin in the blood which is being examined, compared with the percentage of hemoglobin in normal blood. Into the little tube *c* are put a few drops of water to immediately dissolve the blood as it is poured into it. Then a puncture is made with Katsch's scarificator according to the method given above, and, as the blood exudes, by means of the sucking pipette it is slowly sucked up to a mark on this pipette. The point of the pipette is now to be

quickly wiped, and then its contents squirted directly into the water which is at the bottom of the tube *c*. Several times the pipette is to be refilled with water and emptied into the tube *c*, in order not to lose any blood on its capillary wall. The pipette itself may be used for stirring the mixture in the tube *c* if care is taken to completely empty it so as not to lose anything adhering to it.

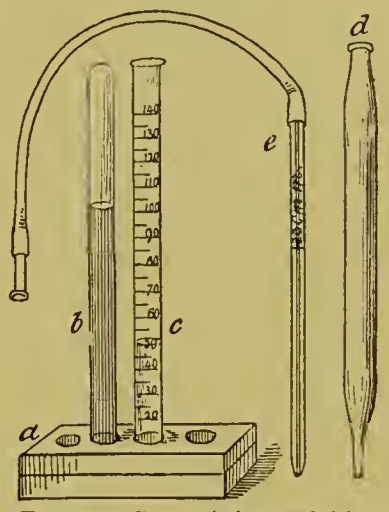


FIG. 82.—Gowers's hemoglobinometer (after Rieder).

Water is now slowly added to the tube *c*, by means of the pipette, till its contents correspond in color exactly with the color solution in the tube *b*. It is best to hold the tubes against a white surface. When the two tubes have been made to correspond exactly in color, the number up to which *c* is filled is read off. The number 100 represents the normal percentage of hemoglobin in the human blood. If the liquid stands at 40, for instance, it means that the percentage of hemoglobin in the examined blood is in proportion of 40 to 100 to that of the normal blood; that is, the examined blood contains only 40 per cent. of the normal quantity of hemoglobin.

From this can be calculated the absolute percentage of hemoglobin in the specimen of blood by putting at 14 per cent. the percentage of hemoglobin in normal blood. The examined blood contains, then, in

$$100 \text{ grams } \frac{40 \times 14}{100} = 5.6 \text{ g. of hemoglobin.}$$

The average limit of error of this instrument is very small: Rieder gives it as 3 per cent. Since its contents become pale with the lapse of time, the test-tube *b* must be renewed from time to time, or it must be controlled by a solution of normal blood which has been diluted to 100. This is not the place to speak of the more exact methods of determining the percentage of hemoglobin. We refer to works upon physiology.

Spectroscopic Character of the Blood.—In certain cases its examination has decided significance. Recently it has been rendered very much more easy by very practical clinical and uncomplicated apparatus, of which we may mention the spectroscope devised by Desaga (Heidelberg), and still more recently Hering's very cheap spectroscope without lenses. The latter, after a little practice, is entirely satisfactory for clinical purposes. The blood or the blood-serum, having been diluted with water, is held in a test-tube before the slit of the instrument and examined against a white light.

In three classes of cases the spectroscopic examination of the blood gives a valuable result; in *hemoglobinemia* there is no doubt about the presence of the coloring-matter of the blood in the serum[1] if the serum shows the absorption-band of oxyhemoglobin; one in yellow near green (close to D, Frauenhofer), and one in green near the former, between D and E. Moreover, in carbonic-oxid poisoning there appear

[1] See p. 232.

in the blood two absorption-bands which are very near the two above mentioned, only a little nearer the violet line, and hence they may be confounded with them, but they are very distinctly separated from bands of oxyhemoglobin in that they do not disappear on the addition of ammonium sulphate (since carbonic oxyhemoglobin is not thus reduced).

Lastly, it has recently been discovered that in poisoning with chlorate of potash, methemoglobin occurs in the blood in the living organism. In acid and neutral solutions this causes an absorption-band in yellow (between C and D), besides three others more faint, which coincide with that of hematin, but which are distinguished from it in that

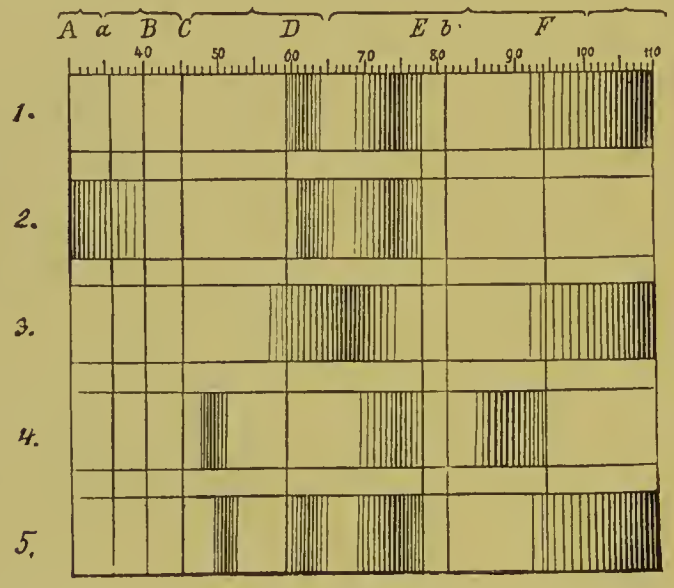


FIG. 83.—Spectrum absorption-bands of the coloring-matter of the blood and its derivatives (after Rieder).

upon the addition of ammonium sulphate it first gives place to the absorption-bands of oxyhemoglobin, then to that of O-free hemoglobin (a broader band from D almost to E in green and yellow). In alkaline solution methemoglobin shows a narrow band in yellow near to D, and one in yellow-green and green.

There are still other changes in the blood, partly relating to its color and partly relating to its behavior in the spectrum, when animals are poisoned, but they do not seem to require special mention in this book.

Determination of the Consistence of the Blood, or its Specific Gravity.—In recent years different methods have been devised by v. Jaksch, Hammerschlag, and Schmaltz. We must abstain from a criticism of them, as we have not made any comparative investigations with them. But we can say of them all that they are superfluous for diagnostic purposes, since Schmaltz found that the specific gravity of the blood is almost exclusively determined by the percentage of hemoglobin; at all events, it goes parallel with it. The determination of the density of the blood can, therefore, be replaced for clinical purposes by the much more simple and comparatively more exact determination of the hemoglobin.

2. Microscopic Examination of the Blood.—The normally formed constituents of the blood are, as is well known, the red and white blood-corpuscles and blood-plates. The morbid conditions of the blood which can be recognized by the microscope may be divided as follows:

Alterations in the number of the red and of white corpuscles, or variations in the numerical proportion of these two components of the blood.

Abnormal size, and form, and peculiarities of the structure of the red cells.

Abnormal quality of the white cells.

Admixtures: These, in the first place, are products of decomposition from the blood itself, and micro-organisms.

About some of these points the fresh, unstained preparation gives explanation; others, and especially the quality of the white cells and most of the micro-organisms, can only be recognized in stained dry preparations.

Mode of Procedure.—For the purpose of making a fresh, unstained blood-preparation there are required object-glasses and cover-glasses, cleaned as thoroughly as possible. If they are cold, they must be warmed a little. Then the place of puncture[1] must be washed with water or a fresh $\frac{1}{2}$ per cent. solution of table salt, and then wiped dry. A puncture is made with a clean needle or with the extreme point of Francke's scarificator.[2] The blood which first escapes is to be wiped off, and that which flows afterward is removed with a cover-glass, which is to be immediately dropped upon the object-glass held in readiness, or a drop is received on the object-glass and covered quickly with the cover-glass without any pressure. It is not advisable to promote the flow of blood by pressure on the parts surrounding the place of puncture. The examination must be made at once, because the red blood-corpuscles, as well as the white, after a short time are subject to alterations. Spots at the edge of the preparation or in the neighborhood of air-bubbles are not to be studied, because here the red cells shrink and decompose. For the purpose of demonstration the preparation can be preserved for a brief time by encircling it with oil.

Counting the Blood-cells.—When one wishes to count the red and white cells a somewhat larger puncture is required. Francke's scarificator, with its lancet arranged so that it comes out one-half its length, is particularly useful here, but in this way a sufficient drop of blood can be gotten from the arm or the ball of the little finger.

Making Dry Preparations.—For this purpose only fine punctures are needed, also very thin, somewhat larger, new cover-glasses, which immediately before being used are to be cleansed with water, alcohol, and ether, and well dried. Take one of them between the thumb and forefinger of each hand; take up with the edge of one some freshly upwelling blood and spread it over the other in the finest possible layer, or pass the other quickly across the spot of the first, which is moistened by the blood.

Microscopical Examination for Micro-organisms.—For this purpose a particularly careful cleansing of the place of puncture and of the

[1] See above.

[2] See p. 232.

glasses is necessary. Strict antisepsis and asepsis is, however, of course, only necessary if the blood is to be used for making cultures.

1. Alterations in the Number and Appearance of the Red Blood-corpuscles.—These are ascertained on the fresh, unstained preparations. A counting-apparatus is required for counting them. The one by far most to be recommended is that of Thoma-Zeiss.[1]

A cubic millimeter of blood from a man normally contains about 5,000,000 red blood-corpuscles; from a woman there are 4,500,000 (C. Vierordt, Laache). A morbid diminution observed in a single examination of a case could only be positively asserted if the enumeration showed one-half of this number or less. The smallest quantity found in disease is about 400,000 to the cubic millimeter.

Oligocythemia is a diminution of the red cells if the whole quantity of blood is taken as the unit of measure. This is the alteration of the blood which accompanies the different forms of *anæmia* (*hydræmia*), of *pernicious anæmia*, and *leukæmia*. Exactly parallel with this may be an alteration in the percentage of hemoglobin; but a complete parallelism may also be absent here, for in pernicious anemia the number of blood-corpuscles is certainly diminished, but the percentage of hemoglobin is greater,[2] and therefore the whole blood contains, it is true, little hemoglobin, but more than would be expected according to the existing oligocythemia. Conversely, in chlorosis the percentage of hemoglobin is much diminished, as has been mentioned before, but there is no, or very little, oligocythemia, because in this disease there exists essentially an impoverishment in respect to hemoglobin.

Counting the Blood-corpuscles.—In anemia, in a stricter sense, it has a diagnostic value, but it has even greater value in that it enables one to recognize the course of an anemia—its improvement or deterioration—and this, after what has already been said in the introduction, forms its diagnostic value in a wider sense. But, as follows from what has been said above, since in chlorosis the number of the red cells has to be considered only a little, and since in common anemias there exists usually also during the course of the disease a certain parallelism between the number of blood-corpuscles and the percentage of hemoglobin, we may say that in chlorosis and simple anemias for determining the course of the disease it is generally sufficient to control the percentage of hemoglobin, which requires less time and trouble than counting the cells.

Method of Counting.—The Thoma-Zeiss apparatus for counting the number of corpuscles is the best of all those now in use.[3] It consists of a mixer and a Hayem's counting-chamber.

The mixer serves to distribute the blood in as equal a manner as possible—a very important point. For thinning the blood a 3 per cent. solution of salt is recommended. The mixer is a kind of measuring-pipette with a very fine canal and with a spherical enlargement containing a little glass ball. The portion of the tube below the cavity has the marks 0.5 and 1.0. Just above the cavity is the mark 101. The

[1] See below.

[2] See below.

[3] Miescher has lately made some alterations in this apparatus which are calculated to increase the accuracy in counting. I have not yet had the opportunity to test the instrument in its new form. It is to be obtained of Karl Zeiss in Jena: Mélangeur after Miescher.

first two marks are those to which the blood, directly after it has been drawn from the finger, is sucked. If we wish a mixture of 1 to 200, we draw it up to 0.5; if a mixture of 1 to 100, to 1.0. In both cases we wash off the blood clinging to the point and draw in a 3 per cent. solution of salt, or Hayem's fluid, to 101. Then the mixer is shaken several times, so that the glass ball equally mixes the contents. We next expel the contents of the fine tube, which consist of salt solution or Hayem's fluid, after which we fill from the mixture a *Hayem's counting-chamber.* This consists of an object-glass with a circular excavation; it is a space exactly $\frac{1}{10}$ mm. deep, the floor of which is divided into microscopic squares whose sides are $\frac{1}{20}$ mm. long. The cubic capacity of the space over each square is $\frac{1}{20} \times \frac{1}{20} \times \frac{1}{10}$ c.mm. = $\frac{1}{4000}$ c.mm.

Hayem's fluid is: hydrarg-bichlorid, 0.5; sodii sulphat., 5.0; sodii chlorid., 2.0; aquæ destil., 200.0.

Into this cavity some of the blood-mixture is blown, and then covered with a glass cover after carefully expelling any air-bubbles.

After waiting a moment, in order that the blood-corpuscles as far as possible may equally distribute themselves, we magnify it about 50 diameters, and count the number of corpuscles in the larger number of the above-named squares, and thus obtain an average of the contents of, say, sixteen of them. The oftener these sixteen squares are counted the greater will be the accuracy of the result. We can calculate the number of corpuscles in a cubic millimeter from the proportions of the mixture and the cubic contents of the squares, as given above.

Immediately after use the mixer must be most carefully washed with water, alcohol, and ether, and it is best to afterward dry it with the air-bellows.

The proportion between the quantity of red cells and the percentage of hemoglobin in the blood is, however, by no means constant, because the percentage of hemoglobin in the individual blood-corpuscles varies in different morbid conditions. This is of diagnostic importance. Here are opposed to each other in a pronounced degree chlorosis and so-called idiopathic or pernicious anemia.

In chlorosis there is a markedly diminished percentage of hemoglobin of the blood, with a slight, or at least proportionately slight, diminution in the number of red corpuscles. The individual cells are even poorer in hemoglobin, as occurs in chlorosis of a high degree in the ordinary fresh microscopic blood-preparation. Dehio has lately found a similar behavior of the blood also in phthisical and carcinomatous cachexia and in the anemia of beginning secondary syphilis (formerly called syphilitic chlorosis), but the investigations of Sadler contradict this.

On the contrary, in pernicious anemia the percentage of hemoglobin is less diminished; the number of red cells, and particularly their whole volume, are more diminished. For this reason the remaining cells are extremely rich in hemoglobin. On the other hand, Dehio found closely similar to this form the anemia caused by the bothriocephalus latus,[1] which also otherwise shows similarities to pernicious anemia or may even change into it.

[1] See below.

2. Alterations in the Size and Form of the Red Corpuscles.[1]—Red cells reduced in size, enlarged, and abnormally shaped may be observed—*microcytes*, *macrocytes*, *poikilocytes*. On all the cells absence of the depression is noticed.

These changes, combined with a decrease in the number of the red corpuscles, and at the same time a normal condition of the white corpuscles, constitute the condition of the blood of so-called pernicious anemia. However, it must here be remarked that lately this is not regarded as a simple disease in itself, since we have learned to recognize it in many cases as a secondary state following different influences very injurious to the body. We shall return to this subject again a little later on.

The simplest way of determining the size is to compare a preparation of blood with that of a healthy person (the examiner himself). The normal diameter of red blood-corpuscles is 7.7 to 8μ [*i. e.* about $\frac{1}{3300}$ of an inch].

Microcythemia.—By this we understand the occurrence of forms containing hemoglobin, which are smaller than red blood-corpuscles, in which the form is nearly or quite perfect, or, if they are very small, they are simply globular, and then are always very rich in hemoglobin. We see the former in the new formations of blood after hemorrhages and also in all kinds of anemia. They are probably young red corpuscles. The latter—microcytes, strictly so called—occur especially frequently in genuine pernicious anemia, and also in all severe secondary forms of anemia. The supposition that they are sometimes formed upon the glass slide is possibly correct, because they may even be found in normal blood if the preparation contains air or if it has been pressed, or also if it has not been freshly made. I have never seen them when examining a perfectly fresh, otherwise normal, preparation of blood, except at the border (the effect of air).

Macrocytes—abnormally large red corpuscles—besides those of normal size and very small ones—occur in individual cases of marked and simple anemia, but especially in pernicious anemia. This disease must always be suspected when they are present. Moreover, very often the poikilocytes to be described below are larger than normal [red corpuscles].

Nucleus-containing macrocytes (*gigantoblasts*—Ehrlich) seem to be the surest sign of a degradation of the blood-making organs and also of pernicious anemia. However, it is to be remembered that, according to our present knowledge, the alterations of the blood in pernicious anemia may occur secondarily to grave injuries of the organism.

Poikilocytes, strictly speaking, are red corpuscles changed in form. They may assume the greatest variety of forms: club, biscuit, pear, flask, and drumstick are the most usual forms. In many ways poikilocytes correspond to enlarged red corpuscles. In individual cases they exhibit ameboid movements. In a wider sense we employ the expression poikilocytosis for a mixture of such forms with microcytes and macrocytes, which are almost always present.

We must avoid confounding with them the mulberry and thorn-apple forms, which occur normally, or mechanical or chemical prod-

[1] Compare Fig. 84, p. 241.

ucts, by using the greatest care in making the preparations and then immediately examining them.

Poikilocytosis, strictly speaking, is not at all in itself a pathognomonic symptom of pernicious anemia, although in other forms of anemia it does not occur so regularly and in so marked a degree as in pernicious anemia. For a diagnosis of pernicious anemia there is necessary the presence of both macrocytes and gigantoblasts (see Fig. 84). As a

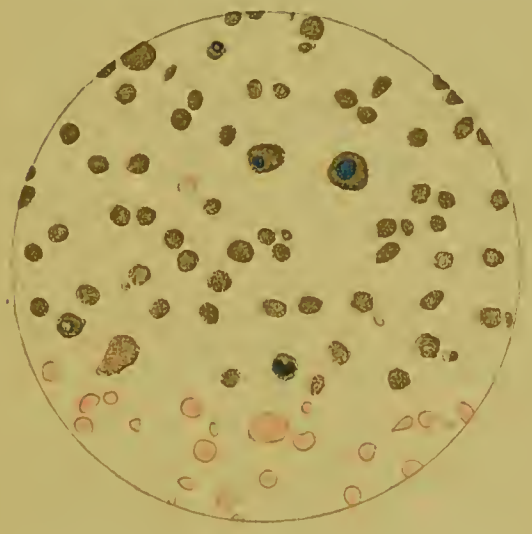


FIG. 84.—Progressive pernicious anemia; magnified 300×. Dry preparation, eosin-hemoglobin. The red blood-corpuscles all without umbilicus. Macrocytes, microcytes, shadows, poikilocytes, two megaloblasts, two normoblasts, one lymphocyte (from Rieder's *Atlas*).

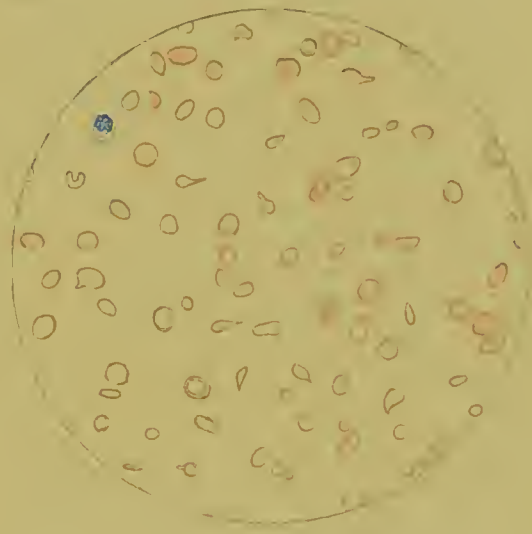


FIG. 85.—Primary anemia gravis; magnified 300×. Dry preparation, eosin-methylene-blue. All the red cells have the umbilicus. Macrocytes, microcytes, poikilocytes (from Rieder's *Atlas*).

distinction from pernicious anemia some call the severe anemia without the presence of megaloblasts "anemia gravis" (see Fig. 85). Pernicious anemia is, however, by no means always an independent disease. On the contrary, it has been observed in connection with tapeworm (especially bothriocephalus latus), with severe leukorrhea, in carcinomatous cachexia, after exhausting hemorrhage from the stomach, and in pregnancy.

As a matter of course, all these changes in the red corpuscles usually very notably accompany diminution in their number and of the amount of hemoglobin. Hence, as has already been mentioned, the amount of hemoglobin in single blood-corpuscles is not infrequently increased.

(Regarding defects within the red corpuscles, which appear in acute infectious diseases and severe anemias, and may be mistaken for malarial parasites, compare under the latter.)

In order to make visible *the nuclei of the red blood-corpuscles* a fixed dry preparation is stained with eosin-carbol-glycerin, and afterward quickly stained with hematoxylin. The method is the same as that employed to bring out the eosinophile white cells. The bodies of the red blood-cells containing the nucleus often appear to have taken up the eosin remarkably strongly.

3. Normal and Pathological Condition of the White Blood-

cells.—The proportion of white corpuscles to the red in normal blood drawn by a puncture, according to the older examinations, was, on the average, from 1 to 400 up to 1 to 700. According to v. Limbeck, more exactly it is as 1 to 555 up to 1 to 625. That is, in a cubic millimeter of blood there are about 8000 to 9000 leukocytes. The white cells of normal blood exhibit different forms: (*a*) mononuclear, partly very small (*i. e.* approaching the red cells in size), cells with so-called basophil-granulation of the body ("*lymphocytes*"); (*b*) polynuclear cells with polymorphic nuclei or with several separated, strongly tingeable nuclei, and finely granulated bodies with neutrophilous granulations; (*c*) coarsely granulated cells with eosinophile granulations and weakly tingeable, often with multiple nuclei ("eosinophile cells"), the latter in very varying number; (*d*) now and then "mast-cells."

An alteration in the proportion of the red and the white cells in favor of the latter indicates either *leukocytosis* or *leukemia.*

In *leukocytosis* the increase of the white blood-cells is more or less temporary and slight in amount. All the forms of the white cells are increased or only the polynuclear neutrophile ones. The latter is particularly the case in the "inflammatory" and in the "cachectic" leukocytosis.[1]

Leukocytosis occurs physiologically during digestion. There is also a so-called "inflammatory" form in acute infectious diseases, particularly in those which are distinguished by the formation of large exudations rich in cells, like pneumonia, but also in erysipelas, pleurisy, and peritonitis. In typhus abdominalis [typhoid fever], however, not only is there no leukocytosis, but, on the contrary, there is a diminution of the white cells. The name inflammatory leukocytosis may also be given to that form which is caused by the swelling of lymphatic glands in all kinds of local inflammation. Finally, there is to be mentioned the cachectic or hydremic leukocytosis occurring in all forms of anemia. This may be a relative leukocytosis, caused by diminution of the red blood-corpuscles, but also an absolute condition, as has been proved by enumeration of the cells. In the latter case it is probably to be explained by the acceleration of the lymph-current, which no doubt exists in consequence of the hydremia.

According to the investigations of v. Jaksch, it appears at least as probable that the number of leukocytes has a prognostic value in pneumonia: he found the inflammatory leukocytosis absent in severe, badly progressing cases.

Leukemia is usually very easily microscopically distinguished from leukocytosis, because in this condition, in well-developed cases, there is a much more considerable increase of the white cells: most frequently there is approximately one white to ten red cells, but in the highest degree the proportion is about one to one.

But in slighter degrees or at the beginning leukemia, especially the myelogenous and lienal-myelogenous forms, may be positively recognized on the basis of Ehrlich's observations. The early diagnosis of lymphatic leukemia, and particularly its distinction from leukocytosis, is certainly more difficult.

In *myelogenous* and *lienal-myelogenous leukemia* there are found in

[1] Compare Fig. 87, p. 243.

the blood: (*a*) one-sidedly increased eosinophile cells—the comparatively least safe sign, because an increase of this formation occurs elsewhere, even in health, particularly in infancy: large eosinophile cells

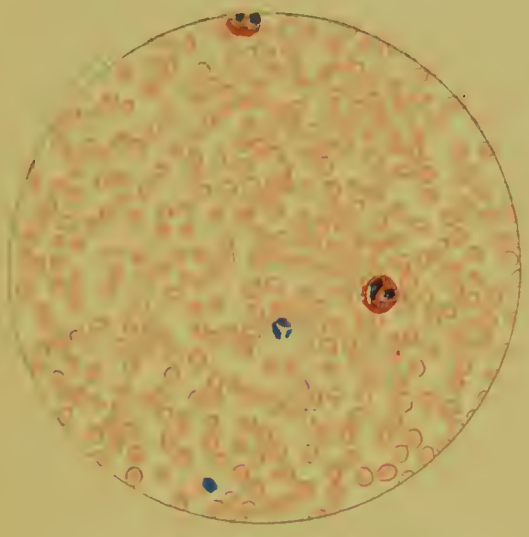


FIG. 86.—Normal blood. Magnified 300×. Dry preparation, eosin-hematoxylin. In the field of vision a lymphocyte, a polynuclear cell, and an eosinophile one. The nuclei of all the white cells dark blue, the eosinophile granulations a brilliant red (from Rieder's *Atlas*).

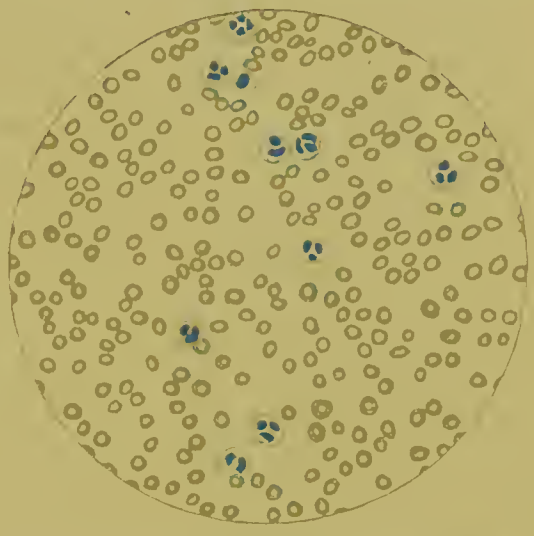


FIG. 87.—Inflammatory leukocytosis; magnified 300×. Dry preparation, Aronsohn-Phillips's staining (see page 245). Marked increase of polynuclear leukocytes. Representation of their neutrophile granules (from Rieder's *Atlas*).

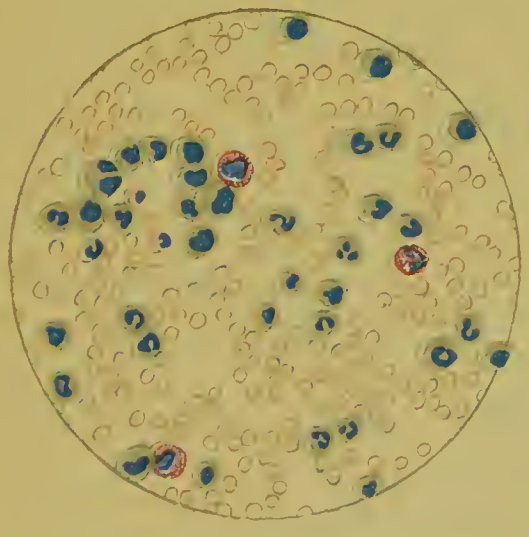


FIG. 88.—Lienal-myelogic leukemia; magnified 300×. Dry preparation, eosin-hematoxylin. Most of the white cells are uninuclear; many are strikingly large, with large plump nucleus. Several eosinophile cells. One nucleus contains red blood-corpuscles (from Rieder's *Atlas*).

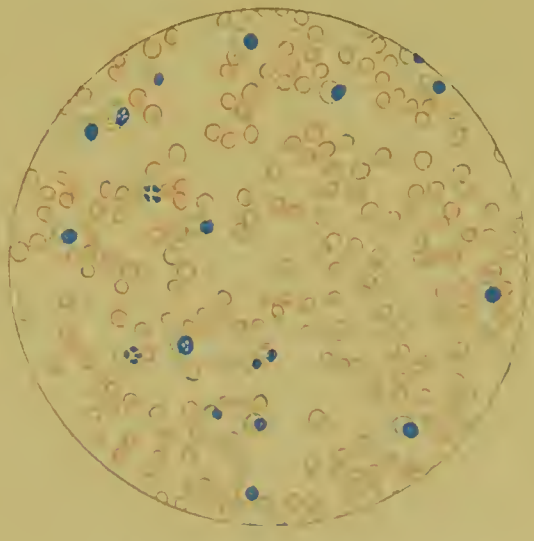


FIG. 89.—Lymphatic leukemia; magnified 300×. Dry preparation, eosin-hematoxylin. Almost all the white blood-corpuscles uninuclear (lymphocytes); most of them very small (from Rieder's *Atlas*).

(marrow-cells) seem, however, only to occur in leukemia; (*b*) very large mononuclear cells, much larger than those of normal blood, and in contrast to those filled with neutrophile granulation-material; (*c*) sometimes abundant "mast-cells," a cell-form which occurs only very

rarely in normal blood; (*d*) nucleus-containing red blood-corpuscles, as large or larger than normal (megaloblasts)[1] (compare Fig. 88).

In lymphatic leukemia, however, there is one-sided increase of the lymphocytes. Thus this form, in slight cases, is similar to leukocytosis, although to the latter belongs either an equal increase of the different forms or an increase of polynuclear neutrophile cells (compare Fig. 89).

Moreover, in leukemia the number of red blood-corpuscles is probably always diminished: v. Jaksch found in making an average of several cases that there are 2,000,000 to 3,000,000 of cells (red and white) to the cubic millimeter.

It is to be mentioned that in leukemia the red blood-corpuscles not infrequently show all the signs of poikilocytosis.

A very rare finding in leukemic blood are Charcot's crystals (probably identical with those of the sputum[2] and feces).

Methods of Counting the White Blood-corpuscles.—For sucking up the fresh blood we employ a mixer having the proportion of 1 : 10 or of 1 : 20 (made by Zeiss of Jena). The diluting liquid is a 3 per cent. acetic acid, which dissolves the red blood-corpuscles. If the red blood-cells have already been determined, the proportion of the white to the red can be quickly calculated. In leukemic blood this proportional number of the white to the red corpuscles may also be obtained by diluting the blood with a 1 per cent. solution of sodium chlorid to which is added some watery solution of gentian-violet. The red and the white corpuscles can then be counted together.

Miescher recently suggested an improved blood-mixer which gives greater exactness in counting the cells (also to be obtained from Zeiss in Jena).

Method of Drying Blood-preparations.—First make very thin cover-glass preparations, and allow them to dry in the air, protected from dust by a bell-glass, or in damp weather in the exsiccator. Then they are fixed either by gradual heating for ten to twelve hours in the drying chamber or on a copper plate at 110–120° C., followed by slow cooling or by putting them for two hours in equal parts of ether and absolute alcohol. If there is need for haste in forming a preliminary opinion, the air-dried preparation may be passed through the flame of a lamp six to ten times.

Method of Staining to Show the Different Cell-forms.—1. *Simple Staining to Demonstrate the Eosinophile-granulations and the Nuclei of White and of Red Blood-corpuscles.*—The dry preparation is to be stained for several hours with a few drops of saturated solution of eosin (bluish, Marke 22, v. Beyer-Elberfeld) in 5 per cent. carbolic-glycerin, rinsing or washing out in water, and restaining with methylene-blue or hematoxylin. In the latter case we use, for a few minutes, Böhmer's or Delafield's solution, diluted with equal parts of water, rinsing in water, drying in the air or in moderate warmth, then sealing up in the usual way. Rieder has even demonstrated mitoses by this method.

2. *Demonstration of the Basophile, Neutrophile, and also of the Mast-cell Granulations.*—(*a*) The *basophile* or δ-granulation of mononuclear

[1] See above, p. 241.

[2] Compare p. 157.

cells (lymphocytes) for several minutes is stained with a concentrated watery solution of methylene-blue. (The solution must always be freshly filtered.)

(*b*) For staining the *neutrophile* or ε-granulation of the common "finely granulated" polynuclear cells we, as well as others, have found Aronsohn-Phillips's modification of the mixture given by Ehrlich to be reliable: a mixture of saturated solution of orange-G extra and crystallized methyl-green extra, after they have been well sedimented, in the following proportions:

Solution of orange,	55.0;
Solution of acid fuchsin,	50.0;
Aq. destillat.,	100.0;
Absolute alcohol,	50.0;

to which is to be added—

Solution of methyl-green,	65.0;
Aq. destillat.,	50.0;
Absolute alcohol,	12.0.

(This is effective after standing one to two weeks; some time later it is less effective again.) The specimens are to be stained for several hours, and then washed out with water, the duration being determined by repeated inspection of the moist preparations; then they are dried, etc.

(*c*) The *mast-cell* granulation is very well stained with Ehrlich's saturated solution of—

Dahlia in absolute alcohol,	50.0;
Aq. destillat.,	100.0;
Acid. acetic. glacial.,	12.5.

It is to be stained for several hours, rinsed with water, and washed out with alcohol for a still longer time, rinsed with water, etc.

4. Abnormal Additions to the Blood.—Of these we first mention melanemia and lipemia.

Melanemia occurs directly after severe attacks of malaria and in malarial disease. We sometimes find, swimming free in the blood, brownish-black or yellow-brown lumps and granules, or also white blood-corpuscles filled with such granules. They result from breaking up of red corpuscles.

By *lipemia* we understand the occurrence of extremely fine drops of fat in the blood, as in drunkards, in diabetes, and in chyluria, but they are also sometimes seen in health.

In recent times we have learned to recognize *micro-organisms* as most important additions to human blood. They are exclusively *schizomycetes*.

About making cover-glass preparations for examinations of micro-organisms, compare page 247. For making cultures it is recommended, in taking blood, to make larger punctures or to take blood

from a vein in which the flow of blood has been stopped (v. Ziemssen) (compare page 251).

Anthrax bacilli in the blood have been repeatedly found in infection by anthrax, although always only in moderate quantity. The defect in the microscopical proof does not exclude, however, a general infection: a test by inoculating mice may, however, succeed.

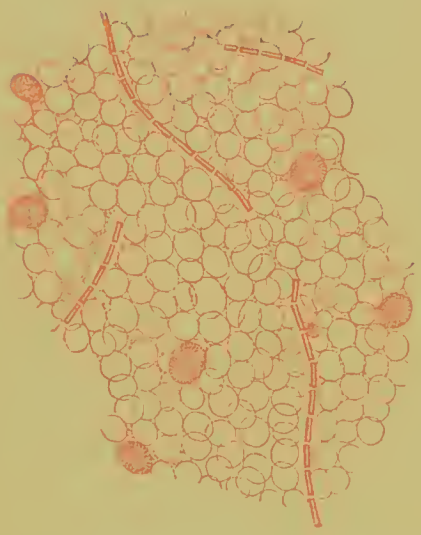


FIG. 90.—Anthrax bacilli in the arterial human blood (fuchsin-staining; Zeiss's homogeneous immersion lens $\frac{1}{12}$, eye-piece 4, camera lucida, magnified about 1000 diameters). The white line in the middle of the bacilli indicates only reflections. Prepared by Dr. Freimuth in Danzig.

We may often have single bacilli of anthrax occurring together, not threads; spores may be entirely wanting. The bacilli are recognized, without staining, as tolerably thick rods, as long as, or twice the diameter of, a red blood-corpuscle.[1]

Spirillum recurrens (Obermeier) was the first micro-organism seen in the blood. We find these organisms during an attack of recurrent fever. They disappear shortly before the decline of the fever. By careful examination they can always be demonstrated, although sometimes only a few of them are found.

In a fresh drop of blood they appear (Hartnack 8, Zeiss F) as extremely fine threads, about five times as long as the diameter of a red corpuscle, with extremely active spiral, serpentine motion. They occur either singly or several close together, sometimes lying together like a group of rats' tails. I have very often first seen them near white blood-corpuscles. The white or red corpuscle against which it lies is usually set slightly in motion by the microbe, and hence we find it there best. Moreover, there often occurs in the blood slight leukocytosis; also, we sometimes meet with shining granules (elementary granules? spores?). As to staining—which, after a little practice, is not necessary—see below.

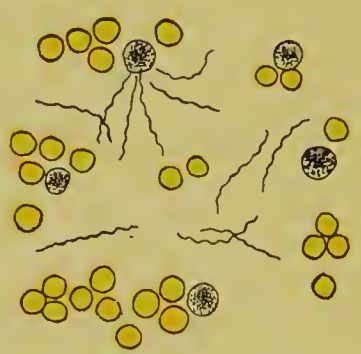


FIG. 91.—Spirillum recurrens in the blood (after v. Jaksch).

Tubercle bacillus exists in the blood as evidence of miliary tuberculosis. But in this disease we may lack this proof. With the exception of one case observed by v. Jaksch it always occurs quite isolated. A special treatment is required for obtaining this microbe. We arrange a thin layer of blood upon the glass cover just as we do a preparation of sputum.[2]

Typhus abdominalis [typhoid fever] *bacilli* have in several cases been found in the blood, taken from one of the roseola spots and from veins, as short (one-third the diameter of the red corpuscle), thick clubs, rounded at the end.[3] (For staining, see below).

[1] Regarding staining, see below.

[2] See Sputum, pp. 159 *ff.*

[3] See Examination of the Stools.

[**Serum Reaction in Typhoid Fever.**—The Widal method of serum diagnosis of typhoid fever has been widely studied, and the more it has been investigated the more favorable have been the results. As modified by Johnson, results have been obtained in 95 per cent. of cases. Others have not had so high a percentage of positive results. Johnson's method is as follows: A drop of typhoid blood is received upon a bit of sterilized paper. This dried blood can be kept at ordinary temperature for a number of days without affecting the subsequent reaction. It is refluidized by a drop of sterile water. This is mixed with a drop of actively motile typhoid culture. The motion quickly stops, and the bacilli run together, forming loose coils or clumps. The time required for this varies from a few moments to several hours—sometimes as many as twenty-four. Incomplete reaction is obtained as early as the second day of the disease, but complete reaction is seldom later than the fifth day. It may take place weeks or months after convalescence, and hence it has been suggested that the Widal reaction is a defensive action—that is, that it is the beginning of an immunity.—*Translator.*]

The *bacilli of glanders* are, in general, a little longer than the preceding, but considerably slimmer. They have likewise been found a number of times in the blood of this disease. It is necessary to stain them.[1]

Mode of Procedure.—The greatest care and cleanliness is necessary in arranging a preparation of blood for microscopic examination for micro-organisms, although the minutiæ of disinfection and sterilization, as in preparing for culture, are not required. In malignant pustule and *febris recurrens* staining can be dispensed with. When it is necessary to stain a preparation, it is prepared by drying a small drop of blood which has been spread out and made as thin as possible by pressing two covers together. Then they are separated, allowed to dry in the air, and afterward passed two or three times through the flame of a spirit-lamp or a Bunsen's burner. If, now, we wish to examine for tubercle bacilli, a special treatment is necessary, as has already been described under Sputum, page 160. For other micro-organisms we stain with basic anilin colors (vesuvin, fuchsin, particularly methylene-blue, etc.), and then carefully rinse and examine in water, or, after drying, in Canada balsam. The staining is much more beautiful if we first briefly dip them in gentian-violet-anilin water,[2] and then stain them a few minutes in Gram's iodin-iodid-of-potassium solution (iodin 1 part, iodid of potassium 2 parts, aq. destil. 300 parts), then in absolute alcohol.

Malaria Parasites.—It is necessary to give a special description of the method of examination for *malaria parasites—hæmatobium malariæ*, wrongly called plasmodium malariæ.

In 1880, Laveran described crescent-shaped bodies in red blood-cells which he found in Algiers in almost all forms of malaria, more frequently in severe ones. In the eighties, Marchiafava and Celli, Golgi, Celli, and Guarnieri studied this phenomenon in Italy. To-day it is

[1] See below.

[2] See above, under Sputum.

established as a fact that in the blood of malaria patients, sometimes in, or more rarely upon, the red blood-corpuscles, again swimming free in the blood, there appears a parasite, of exceeding variety of form, which is never found in the blood of any other patients, and which finally disappears after the patient has taken sufficiently large doses of quinin. The parasite is found at the time of the fever, or its appearance in the blood precedes the fever. In slighter forms of malaria (febris quotidiana, tertiana, quartana) there seems to be no possible doubt that a new generation of the parasite in the blood corresponds with an attack of fever. The parasites of the severe forms of malaria, existing only in warmer countries, and those of the febris intermittens, which have alone to be considered in Germany [England and the United States], do not seem to be entirely identical.

Method of Procedure.—A drop of blood is taken from the cleansed finger-tip or the lobe of the ear in the manner already described; the cover-glass is strongly pressed upon it; the preparation is protected from evaporation by surrounding it with wax. It is strongly to be recommended, after the method of Celli and Guarnieri, to mix the fresh drop of blood during its exit on the finger-tip with methylene-blue serum.[1] In the course of a half to one hour the leukocytes and the malaria parasites are stained, the latter very distinctly. *Dry preparations* must be spread very thinly, are not to be passed through the flame, but are air-dried, fixed with absolute alcohol, in which they must remain about ten minutes, and dried again in the air. It is best then to employ a solution of eosin-methylene-blue (Plehn): equal parts of concentrated watery solution of methylene-blue; water; 60 per cent. alcoholic solution of eosin. Staining is continued from one to twenty-four hours, rinsed in water, etc. The solution must be filtered before using. It stains most intensely when two to eight days old, after which period its staining is less intense. Mannaberg[2] has lately recommended as the best a method proposed by Malachowski. The fluid consists of the following:

Concentrated watery solution of methylene-blue,	24;
5 per cent. solution of borax,	16;
Water,	40.

After standing twenty-six hours it is to be filtered. Specimens are to be stained in this for twenty-four hours, and then washed off in water. (Sometimes a few granules of eosin are added to the fluid.)

The parasites appear as pale lumps of protoplasm which generally, but not always, enables one to recognize a differentiation in their substance: lying within darker parts of protoplasm there are circumscribed lighter (also paler-colored) parts ("endoplasma"), which give the impression of vacuoles. These latter, in preparations stained after the method of Plehn, shine through the eosin color of the blood-corpuscle stroma so intensely that the darker "ectoplasma" appears like a ring (see Fig. 92, 1 and 2). The larger formations contain red-brown or brown-black, nearly round or rod-shaped pigment-granules. Sometimes the

[1] Concentrated solution of methylene-blue in sterile serous transudation-liquid.

[2] *Fortschr. der Medicin*, 1893.

parasite appears in the form of a glomerular frame in the interior of the blood-corpuscles (4); rarely, in temperate climates, to appear in the form of a crescent (8); rare also is the so-called star-flower form of the protoplasm (5). The bodies swimming freely in the plasma (7) are dim

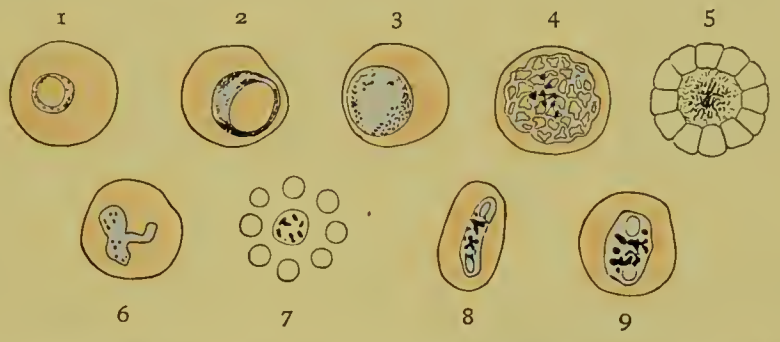


FIG. 92.—Malaria parasites (eosin-methylene-blue. 5 and 7, unstained preparations).

1, 2, 3, 6, two ring-like, one ball-shaped, one branched, plasmodium; 4, glomeruli form; 5, aster form, beginning segmentation; 7, complete segmentation; Laveran's crescent; 8 and 9, show vacuoles (partly after Dolega, partly after Quincke; magnified about 1000×).

disks of the size of a red blood-corpuscle or smaller. In the fresh preparation the smallest sometimes appear like whip-threads.

The parasites, similar to the leukocytes, in the fresh preparations show ameboid changes in form, which are somewhat quickened by warmth. Their pigment-granules are sometimes in lively motion, partly currents in the protoplasm, partly molecular movements.

Later investigations seem to confirm an opinion expressed by Golgi, that different forms of the plasmodium correspond to different types of intermittent fever. Likewise, it seems to be certain, as has been mentioned above, that other, smaller, forms of the parasites correspond to the severer malarial fevers as they occur, for instance, in Rome during summer and autumn.

In *black-water fever* there have lately been found formations which very much resemble the malaria parasites.

At the same time with the malaria parasites there is a moderate *leukocytosis*, comparatively many eosinophile cells, and probably also leukocytes which enclose plasmodia. But, besides that, there is a very remarkable *alteration in the red blood-corpuscles*, which may cause, and has already caused, them to be confounded with the parasites: individual red corpuscles in their interior show enclosures which appear as colorless or very palely-colored circles or clubs, ellipses, etc. They may lie concentrically, and are without doubt to be taken for deepened, sometimes also for steep-walled, dells, by which dells the stroma of the respective blood-corpuscles is made thinner, partly also by the disappearance of its substance. These *pseudo-vacuoles* (Quincke; "vacuoles," v. Jaksch) are distinguished from the malaria parasites by being sharper circumscribed and of course free from granules, and that they are not stained by methylene-blue and do not show any ameboid movement, but frequently another—*i. e.*, as it were, a pulsating movement—probably conditioned upon the ascending and descending of the blood-corpuscles in the liquid layer. They also change their form from the circular into the ellipse form, etc.

This alteration of the red blood-corpuscles seems to be a simple analogue of poikilocytosis. It is observed not only in malaria blood, but also in that of measles, scarlet fever, typhoid fever, ephemera, likewise in anemic patients, in carcinosis, scurvy, leukemia, and now and then in the healthy.

Finally, we briefly refer to two animal parasites which are met with in the blood (compare Figs. 93, 94), though they do not belong to our climate: the *filaria sanguinis hominis*, which causes hematochyluria



FIG. 93.—Distoma hæmatobium with eggs (after v. Jaksch).

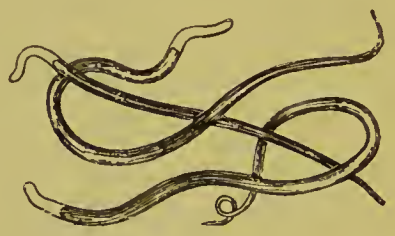


FIG. 94.—Filaria sanguinis hominis (after v. Jaksch).

(in British India and Brazil), generally only found in the blood at night-time, and *distoma hæmatobium* (Bilharz), which causes a kind of hematuria, chiefly occurring in Egypt.[1]

Chemical Examination of the Blood.

We content ourselves with a few hints regarding this department, which in recent times has been much elaborated.

The interesting investigations made by von Jaksch regarding the percentage of nitrogen and albumin in the blood, by Stintzing regarding the percentage of water, and, most important of all, that the percentages of albumin and water are always in an inverse proportion, are topics which lie outside of the domain of diagnosis.

Recently, in certain diseases, *the degree of alkalescence* of freshly-drawn blood has been determined by various methods, and it has been found that in severe anemia, fever, and diabetes (v. Jaksch), in decomposition of red blood-corpuscles, the alkalescence is considerably diminished. These investigations are not to be valued very highly, because their results are impaired by great technical difficulties. *Uric acid* in unusual quantity has been found in the blood in gout. It is also easy to demonstrate the amount of bile-pigment and urobilin in the blood and the amount of hemoglobin in the serum if the quantity of blood is sufficient—at least 3 c.cm. (Tissier, v. Jaksch).

The quickness with which blood coagulates after it has been withdrawn varies in different diseases. In health coagulation takes place in about nine minutes. It is slower than this where the nutrition is chronically disturbed (H. Vierordt).

Puncture in the tip of the finger or some other place, however, does not generally furnish the requisite quantity of blood for these examinations. They require somewhat larger quantities, which till

[1] See under Urine.

now have been obtained by means of a scarificator and cupping-glass or by venesection. A short time ago v. Ziemssen proposed a method for obtaining larger quantities of blood, which in consequence of its harmlessness and exactness may be substituted for the two last-named methods. With the necessary asepsis blood is taken from the median vein on the forearm after having stemmed the blood by light compression on the upper arm. The blood is removed by a small aspirating syringe whose needle is thrust into the vein in a distal direction and pushed in parallel to the skin. In this way, at discretion, smaller or larger quantities of blood may be drawn, and the procedure may also be repeated at the same place.

CHAPTER VI.

EXAMINATION OF THE DIGESTIVE APPARATUS.

MOUTH, PALATE, AND PHARYNGEAL CAVITY.

The Mouth.—The *inspection* of these parts requires good illumination, and for a portion of them, in many cases, a quick view. Bright daylight is better than artificial light. The mouth is to be opened widely, the tongue protruded, and not only put out, but, for inspecting its borders, turned from side to side. (For examining it with reference to paralysis, see Nervous System.) In order to inspect the mucous membrane of the mouth, we turn out the upper and lower lips with the finger, the mouth being closed; then, the mouth being opened, we carefully lift the mucous membrane of the cheeks from the back teeth with a mouth-spatula (made of ivory, hard rubber, horn, or metal [best of all, glass]). The gums are examined by opening the mouth as widely as possible and holding the tongue down carefully with a tongue-depressor (a teaspoon serves very well). The back of the mouth is best brought into view by having the patient say distinctly *ā* (full elevation of the soft palate). Often the patient must be required to drink some water, or to clear the throat thoroughly by hawking before it is examined. If we meet with opposition, especially in children, it is sometimes necessary to hold the nose, and thus compel them to open the mouth. When a child cries, we are able to see very well. It is often useful to cause the sensation of strangling by putting the tongue-depressor far back, and thus we are able to see the tonsils better—of course, only for an instant. [One learns, by practice, to take a very perfect and complete view of the whole cavity of the mouth and pharynx in this instant of strangulation, and then can carry the mental picture long enough to note all its particulars.]

But we must guard against being too harsh or rough with children with *diphtheria*, or with any very sick patient. In diphtheria immediate death may follow an effort at examining the throat. With those who are unconscious it is necessary to cause gagging in order to inspect the posterior part of the mouth. In marked cases of this character it is often impossible to obtain a view at all.

Palpation is only rarely employed for examining the tongue, floor of the mouth (making counter-pressure from without), the tonsils, or the back part of the pharynx. We employ the index or this and the middle fingers, which have been carefully washed in the presence of the patient.

The odor from the mouth is, in many cases, important. A foul odor —*fœtor ex ore*—results from imperfect cleansing of the teeth, caries of the teeth, or dyspepsia. From this odor we distinguish the stale, and

at the same time foul, fetor from considerable old deposit in the mouth of patients who are very ill. If the sense of smell is acute, one can also distinguish a slight cadaveric odor upon patients who are very sick, even if the mouth is quite clean, and sometimes it precedes death.

Of much more diagnostic value are the different *odors* which we meet with *in poisoning* from prussic acid, phosphorus, alcohol, and chloroform; but the former two, even in recent cases, may possibly be wanting. Lastly, we mention the odor of fruit, wrongly called "acetone" odor, very like fresh apples, which sometimes occurs with the so-called chlorid-of-iron reaction of the urine[1] in diabetes, especially before or during the onset of diabetic coma, or during its course, as well as in other conditions.[2]

The Lips.—With regard to their color (pale, cyanosed, etc.) we can refer to what has already been said when speaking of the mucous membrane. Dryness of the lips is seen in connection with dryness of the tongue.[3] There is marked dryness in severe febrile diseases, with a dirty-looking crust adherent to the mucous membrane, which easily bleeds when this is removed (fuliginous deposit). Small cracks (rhagades, crevices) are, in themselves, without significance. On the contrary, in children rhagades are an important, generally a positive, sign of hereditary syphilis.

In persons affected with hereditary syphilis, in rare cases, one sees, besides, peculiar deep wrinkles in a radial position round the mouth. They particularly run toward the corners of the mouth in the manner of a *pes anserinus*, but occasionally they also go as from the chin to the lower lip. They might be taken for linear scars were not their origin in corresponding long and deep rhagades excluded. I have seen them in children a few months old, as well as in adults, but exclusively in hereditary syphilis. Their origin is not at all clear.

The Teeth and Gums.—We must take their condition into consideration together, and, besides, as to whether the teeth are sound. In small children we notice whether the first teeth have all come; in the later years of childhood, the change to the permanent set.

There is often marked *caries* of the teeth in *diabetes mellitus*, though it is very common without this disease. A circular excavation of the lower edge of the upper middle incisor teeth of the second dentition [Hutchinson's teeth] sometimes occurs, although it is a very uncertain sign of congenital *syphilis*, with catarrh of the middle ear and parenchymatous keratitis, the whole forming the so-called Hutchinson's triad—a group of symptoms which very seldom present themselves, whose significance in the diagnosis of hereditary syphilis does not seem to us so infallible as was formerly supposed. Imperfect and diseased teeth, interfering with mastication, are often the chief cause of dyspepsia.

Loosening of the teeth, and the gums discolored bluish-red, receding from the teeth, easily bleeding, and even inflamed, are important symptoms of *scorbutus*. Loose teeth, with moderate swelling, is a sign of chronic poisoning with mercury.

A grayish deposit upon the teeth, and especially a gray line along the dental border of the gums, are of importance for the diagnosis of

[1] See chapter on Urine.

[2] See chapter on Urine.

[3] See below, p. 254 *f.*

chronic lead-poisoning. In poisoning by copper we have sometimes the same condition, only the color is greener.

The *eruption of the first teeth* is a source of much disturbance in the mouths of the little patients. Occasionally it gives rise to serious disturbances—diarrhea in rare cases, epileptiform attacks (eclampsia of children, infantile convulsions, spasms of dentition), also spasm of the glottis. Also, *second dentition* and the eruption of the wisdom teeth are not infrequently accompanied with limited or general oral disturbances, sometimes likewise the cause of abscess.

To the red border upon the gum, observed by Frédéricq-Thompson, which in young subjects is said to be a very suspicious sign of tuberculosis, we have given careful attention for a long time, and conclude that it has no significance.

The Tongue.—For paralysis and neurotic atrophy of the tongue, see under The Nervous System.

Enlargement of the tongue, if slight, is only to be determined from the indentations on its borders by the lower teeth. This occurs with the various forms of *stomatitis*. Marked enlargement of the tongue may be caused by parenchymatous glossitis, tumors, and also by severe angina, which produce venous engorgement of the tongue. Moreover, there are very great individual variations in the size of the tongue.

Circumscribed swelling and *hardness*, or the latter alone, are the first evidences of carcinomatous or syphilitic formations of the tongue. It is extremely difficult to make the very important differential diagnosis between these new growths, and usually it can only be made *ex juvantibus*, or by microscopically examining a small piece, which can easily be removed from it.[1]

Wounds and the resulting *scars*, sometimes accompanied with swelling, are frequent appearances after epileptic attacks and result from biting the tongue.[2] We never see the tongue bitten in *hystero-epilepsy*.

If the *tongue trembles* when it is protruded, or if it does so when within the mouth, it is a valuable sign of chronic *alcoholism*. This is also the case in severe fevers, and especially early in typhus abdominalis [typhoid fever]. In these cases, when there is marked hebetude, the patient often will not draw in his tongue after protruding it unless he is directed to do so.

The *color of the tongue* is affected by that of the blood: cyanosis affords the most marked instance of this. It is quite common to find local redness with febrile conditions. It often goes side by side with the febrile redness of the cheeks. Mulberry tongue is one in which there is a decided redness with swelling of the papillæ, and is an important sign of scarlet fever, which in individual cases may develop before the cutaneous eruption. Very often the coating of the tongue conceals the color of the mucous membrane.

When the saliva is glutinous or diminished it causes the tongue to be sticky or dry. In connection with dryness of the throat febrile diseases cause thirst. When the fever is very high the dryness is often increased by the patient keeping his mouth constantly open. Then the

[1] Regarding this, see works upon Surgery.

[2] See under Scars.

surface of the tongue, if free from coating, first becomes horny, then quickly very smooth, and soon rough and cracked.

Coating of the tongue as a thin white layer is often constant in health. When a tongue which previously was clean becomes coated, especially if thickly coated, it indicates dyspepsia. There is very marked coating of the tongue in severe acute and chronic diseases of the stomach and with the dyspepsia of fever. With the latter it is often discolored brownish-red from small hemorrhages of the mucous membrane. When there is great dryness of the tongue it becomes covered with crust, which adheres so closely that when removed the mucous membrane bleeds. Articles of diet may cause temporary coating or they may color the coating that is already there (milk, cocoa, coffee, etc.).

A thick white—often also a discolored—coating on the tongue may depend upon the development of *thrush* (oïdium albicans). In very pronounced cases it forms separate small tufts about the size of a millet-seed, which spread out and coalesce. It is cheesy and tolerably adherent. It may cover the surface of the tongue, the soft and hard palate, the mucous membrane of the cheeks; it may even extend down into the esophagus; occasionally we see the whole surface of the mouth and throat covered with it. [The translator has seen a few cases where it seemed to have spread the whole extent of the alimentary tract, appearing about the anus.] Small children have it quite often, adults only in cases of severe illness when the care of the mouth is neglected, especially in fevers, diabetes, tuberculosis, etc. Whenever there is a thick coating in the mouth we must think of this growth, because its early recognition is very important. The diagnosis is promptly made by the aid of the microscope.[1]

For scars from biting of the tongue during an attack of epilepsy, see above, under Wounds. Dense, often depressed, scars upon the surface of the tongue indicate healed syphilitic ulcers.

In tertiary syphilis there is not infrequently a peculiar smoothness of the mucous membrane of the back of the tongue at its root, which is to be explained by an atrophy of the mucous membrane, and particularly of the follicles of the tongue. This alteration may be perceived by palpation and by examination with a mirror.

Mucous Membrane of the Mouth.—When there is a suspicion of *syphilis* the mucous membrane of the mouth must be examined with the greatest care (scars, ulcers [mucous patches]); also when there is a possibility of poisoning with strong mineral acids or alkalies, corrosive sublimate, carbolic acid (superficial gray color, and under it marked injection of the mucous membrane, raw patches). It may also be the seat of *catarrhal ulcers* as well as of the development of thrush.[2] *Cancrum oris* (noma) usually begins with a circumscribed bluish-black discoloration of the mucous membrane of the cheek or an ulcer with this condition around it, and with a thick, inflammatory infiltration of the cheek. It is a kind of spontaneous gangrene with a decided reactive inflammation in poor, wasting children. It is a rare disease.

We examine the floor of the mouth by palpation from within and without. It may be the seat of very dangerous inflammation (*angina Ludwigii*).

[1] See next page.

[2] See below, also above.

Salivary Glands and Saliva.—Of the former we notice only the parotid gland. When it is inflamed there are pain and swelling, and if it proceeds to the formation of an abscess, there are also redness and fluctuation above the angle of the jaw.

The saliva is *increased* (salivation, ptyalism) by all kinds of irritation that affect the mucous membrane of the mouth—physiologically by eating, pathologically by all inflammatory conditions of the mouth (ulcers, inflammation of the gums in connection with affections of the teeth, dental abscess, etc.; corrosive action of acids, alkalies in the mouth and throat); also, in chronic mercurial poisoning: and, lastly, sometimes in disease of the medulla oblongata.[1] The saliva is *diminished* in febrile diseases, in diabetes, in severe diarrhea (cholera). Thus far, the chemical examination of the saliva has been of no diagnostic value. It is of interest that in *nephritis* it may contain urea, and also that thus far there has never been discovered in it any coloring matter from the bile, nor any sugar. Many substances, like iodid of potassium, after they have entered the stomach appear remarkably quickly in the saliva.

Microscopic Examination of the Contents of the Mouth.—Normally, we find flat epithelial cells from the upper layer of the



FIG. 95.—Leptothrix buccalis, from the sputum of a phthisical patient. Fresh specimen treated with lactic acid and Lugol's solution. Magnified about 300×.

mucous membrane of the mouth, separate white blood-corpuscles, and likewise micrococci, bacilli, and spirocheta in great abundance. Among these are very long threads of variable thickness, often arranged parallel, and resembling a tuft of hair. They are especially found in the material adhering to the teeth. They take a brilliant violet color if the preparation is a little acidulated with 5 per cent. solution of lactic acid and a drop of Lugol's solution is added. These

[1] See Bulbar Paralysis.

formations have formerly been described as a single fungus, *leptothrix buccalis*. It has, however, been asserted that different bacilli in the secretions of the mouth are peculiar in that they grow out in long threads and give the iodin reaction. It is certainly a striking fact that some short portions become stained by iodin—some just as much and some a little lighter than the leptothrix. Nevertheless, and although certain results of culture furnish ground for the opinion, we do not think the point has yet been proved. We rather think there is reason to believe in the oneness of character of the so-called leptothrix threads which occur in the mouth (and in the tonsils and sputum).[1]

Of considerable importance is the appearance of *pathogenic microorganisms in the buccal secretion of healthy persons*. From it there have been produced by culture the bacterium of rabbit septicemia, the sarcina-like micrococcus tetragenus (it has been alleged also), actinomyces (?), a diplococcus similar to Fränkel's, the bacillus of diphtheria, and lastly strepto- and staphylococci.

Furthermore, some bacteria of the mouth show a striking resemblance in the culture to certain pathogenic fungi, without being identical with them, as, for instance, a bacterium which is similar to the comma bacillus and one similar to that of the recurrens spirochæta.

Again, there are found a great number of non-pathogenic bacilli and cocci.

In general, the bacteria of the mouth are increased in all forms of *dyspepsia;* they are found in very great quantities in persons suffering from any severe disease, and in all forms of stomatitis.

Two diagnostic points have yet to be emphasized:

It is easy to recognize the *thrush-fungus* under the microscope by the characteristic, tolerably broad, light fungus-threads (they are more than half as broad as a white blood-corpuscle) and by their roundish-oval, clear granules.

Suppurations in the mouth proceeding from the inferior maxilla are, in rare cases, caused by *actinomyces*. Whenever there is a discharge of pus into the mouth we must remember the characteristic granules.[2]

The soft palate, the uvula, palatal arches, and tonsils are the seat of diseases of the most different kinds. These parts, also, are exposed to the greatest variety of injuries, particularly to infections, since not only the nourishment which is brought to the body passes along them, but also the respiratory current of air, and since they probably also participate in infections which are taken up from the mouth-cavity, principally by kissing. In addition, there is a local disposition to the absorption of infectious germs, which disposition is without doubt peculiar to the tissue of the tonsils. Thus all possible kinds of pathogenic cocci—diphtheria bacilli, syphilis-poison, tubercle bacilli, leptothrix, actinomyces—find lodgement in the palate or its immediate surroundings. There are also chemical injuries—*i. e.* corrosions in poisoning with strong acids and alkalies—and mechanical ones by fish-bones, bone-splinters, puncturing the tissues.

Of some infectious diseases not mentioned here it is still unproved, but probable from clinical reasons, that they obtain their entrance through the tonsils. In scarlet fever this is an extremely probable

[1] See pp. 150 and 263.

[2] See p. 139; Microscopic Examination, see p. 150.

supposition; but it occurs also in measles, in some cases of articular rheumatism and typhoid fever, in certain infectious forms of nephritis.

In a manifold way the tonsils, moreover, are the seat of origin of a septico-pyemic general disease; that is, either by the immediate primary absorption of pyogenic material (tonsillar abscess as a cause of septicemia) or by secondary infection in the tonsillar tissue which has been prepared by some other primary disease. One must think of the secondary invasion of pus-cocci in bacillar and in scarlet-fever diphtheria. The form and color of the individual parts of the palate must be carefully observed. An exact knowledge of the normal appearances is of course indispensable for a correct judgment of pathological conditions.

As regards the *general rules for the diagnosis* of these different morbid states, the directions which we have given on page 252 must be carefully observed. It is particularly desirable to examine difficult cases, which are notably frequent, by daylight or in an artificial white light which most nearly resembles daylight. The form and color of every part of the palate must be observed. An exact knowledge of the normal conditions, as has been said, is of course indispensable for a correct judgment.

Special diagnosis cannot of course be here entered into exhaustively. We take only the most essential points:

Chronically hypertrophied tonsils are to be distinguished from freshly inflamed organs by their normal color and insensibility. Wide and deep empty lacunæ point to frequent attacks of angina [tonsillitis].

Secondary syphilis produces on palate and tonsils condyloma-like plaques—*i. e.* broad, flat, sharply defined, whitish patches, or simply reddened spots which are striking by their sharp demarcation and a never-failing, although small, elevation. Finally, there are also seen flat or deeper, always sharp-edged, ulcers with gray-colored edges. *Tertiary syphilis* produces more solid infiltrations, with ulcers of quite different depth, covered with yellow purulent matter. From this cause also there may be great defects of the palate. *Syphilitic scars* are recognized by their sharply distinct white lines on the tonsils, often by simultaneous deep retractions. A favorite seat of these scars is the upper part of the posterior palatal wall, which becomes visible only in pronouncing *ā*.

The *acute anginas* form a group of cases of great variety. In the foreground of interest stands the question as to how clearly the forms associated with genuine exudations can be distinguished from one another and from the simple lacunar and necrotic anginas. The question which arises most frequently is regarding a differential diagnosis between bacillar diphtheria and lacunar angina [follicular tonsillitis]. Mistake is impossible in fully-developed *diphtheria*. The strongly adhesive, whitish exudation adheres to the tonsils as a solid membrane, spreads in irregular form over the neighboring tissues on to the soft palate and farther into the buccal cavity. Also, in the beginning of diphtheria the distinction can often be made with certainty, because even the small, closely adherent exudations, irregular in shape, are distinguished from the plugs of lacunar angina [follicular tonsillitis], the one variety being closely adherent and irregular in shape, while the other

can be readily detached from their lacunæ, and are round, slightly protruding, yellow masses. But sometimes diphtheria commences likewise in the form of small, roundish, plug-like formations scattered on the tonsils. If viewed closely, they often show suspicious peculiarities: they are strikingly whitish; they do not rest in the lacunæ, but on prominent spots of the tonsils; further, they occasionally resemble nail-cultures—*i. e.* they very early show a delicate membranous superficial surface around the plug, resting in its lacuna. But occasionally these signs fail: the angina certainly seems to be lacunar [follicular tonsillitis], but the bacteriological examination and the further course proves it to be diphtheria. I have directed my attention to these things for years, and am obliged to strongly take issue against Sahli that these happenings are not too rare.

Lacunar angina [*follicular tonsillitis*], which occurs during epidemics of diphtheria, or, worse still, in families, houses, and schools, ought immediately to be examined bacteriologically and isolated.

More difficult still is the early distinction of diphtheria from *simple necrotic angina*, which sometimes occurs as a primary disease, and is then usually unilateral, or as a primary lacunar angina. The exudations of these forms adhere less closely than the diphtheritic, and they do not extend beyond the tonsils; but these are of course uncertain signs. Here, too, it is well to examine bacteriologically.

Finally, there is the differential diagnosis of diphtheria from the other forms which are associated with exudations.

Scarlet fever is often distinguished by a gray, dirty, or very delicate membrane; in other cases it strikingly resembles bacillary diphtheria, with which etiologically it has nothing to do. The diagnosis is clear if the complex of symptoms of scarlet fever is distinctly pronounced. It must be observed, however, that in bacillary diphtheria complicating scarlet-like exanthemata may be caused by sepsis—that, *vice versâ*, in scarlet fever the exanthema may be rudimental or even entirely absent. An extension to the larynx always points to bacillary diphtheria.

There are, besides, some peculiar diphtheroid *anginas caused by strepto- and staphylococci* which have nothing to do with scarlet fever. These generally have an entirely different appearance from diphtheria: the coating is gray or yellowish, delicate and soft, frequently, however, as sharply circumscribed, as decidedly adherent, as the genuine diphtheritic membrane. They may also extend beyond the tonsils. These affections are certainly transmissible, but they remain confined to the palate and the prognosis is favorable. They have, therefore, to be separated from diphtheria, not only etiologically, but also for other reasons. But it is principally these affections, besides scarlatinous diphtheria, which have led many authors to speak of a genuine diphtheria without bacilli, and to doubt the etiological significance of Löffler's bacillus.

We recognize an *abscess of the tonsil* by its [usually] being on one side only, with swelling of the anterior arch, by the fluctuation (which is felt with the finger).

Long-continued *ulcers of the tonsils* and soft palate are generally *syphilitic*, more rarely *tubercular*. In the latter case there are often, besides, larger ulcers, or also, without them, a broad, reticulated, puru-

lent discoloration of the mucous membrane, which reminds one of slightly-inflamed pleura covered with a fine fibrinous exudate. For paralysis of the throat—see Nervous System.

In the pharynx we look for possible chronic or acute inflammation and ulcers; in *children* who, for some unknown reason, swallow badly and have distress in breathing, for possible swelling of the posterior pharyngeal wall (*retropharyngeal abscess*, the fluctuation in which may be detected by palpation).

We must always examine the *lymphatic glands* in the neck in connection with the examination of the throat. In all acute inflammations of the latter they swell, most markedly in diphtheria, also in chronic inflammations, especially in syphilis.

From what has been said above the inference will readily be drawn that after much practice and large experience, especially by training the eye to make sharp distinctions and by becoming familar with the normal and the pathological pictures, one may be able by simple inspection to make a very close approximation to an accurate diagnosis of the affections of the palate; but, nevertheless, it is frequently necessary to employ the bacteriological examination, especially when it is a question as to the existence of diphtheria or tuberculosis.

Microscopical and Bacteriological Examination of the Palate and Pharynx.

When **tuberculosis** of the palate and pharynx is suspected, by means of a flexible spatula, such as is used in microscopy, we scrape some particles from the bottom of the ulcers, and make cover-glass preparations, which are to be examined for tubercle bacilli like sputum-preparations. As these patients always suffer also from tuberculosis of the lungs (or larynx), it is necessary to be careful not to get any sputum, which adheres accidentally to the mucous membrane, upon the spatula. For this reason it is desirable to have the patient gargle before commencing the manipulations.

Bacteriological Diagnosis of Genuine Diphtheria.—In every case of necrotic angina or of angina with exudation which is in the least doubtful one must examine for diphtheria bacilli. The conscientious physician, who regards the danger to which children are exposed, will also subject lacunar anginas to this examination. The reason for this is clear from what has been said above. At the present time we are positive that genuine diphtheria is always and only produced by Löffler's diphtheria bacillus; the cause of simple necrotic and simple lacunar angina we do not yet know. The cause of scarlet-fever diphtheria is either the yet unknown, peculiar scarlet-fever poison, or a chain coccus, which is never absent in scarlet-fever diphtheria, and may also extend from this into the surrounding tissues, the glands, and the circulation. At any rate, the genuine Löffler's bacillus is also found in convalescence from diphtheria, and it has even been found in isolated cases in healthy persons (Löffler, Babes, v. Hoffmann); but that will scarcely interfere with the differential diagnosis in a case of doubtful acute angina [follicular tonsillitis]. On the other hand, it is of greater significance that in microscopical examinations the bacillus may be easily confounded with

other similar ones: in the diphtheria membrane it neither has always a characteristic appearance nor has it a specific coloration peculiar to itself. Therefore, *the exact proof of the diphtheria bacillus can generally only be furnished by culture.* If the specimen is spread on an agar plate, a reliable result in a positive case can be obtained in twenty-four hours. It rarely requires more time for the development of characteristic colonies, and also there are rarely so many different bacteria that a second plate is needed. Likewise, it is seldom necessary to make a bouillon culture, as the bacilli develop very typically on the plate.[1]

The vaccinating experiment can almost always be dispensed with for the purposes of diagnosis.

However, the simple *microscopical examination* of a piece of membrane, according to our experience, sometimes gives an immediate positive result, and it should never be omitted in a doubtful case; for if in the pieces of membrane there are found oblong bacilli in heaps or only in nests, or even if they are found only here and there quite distinctly, without admixture of cocci, etc., we have always to do with diphtheria. If, however, only isolated specimens of Löffler's bacillus are found mixed with other bacilli and cocci, no conclusion can be drawn from the preparation. Culture alone can decide.[2] Our examinations have shown that just in the beginning of the disease the microscopical preparation sometimes does not permit a reliable conclusion; and this is certainly often annoying, for an early diagnosis is

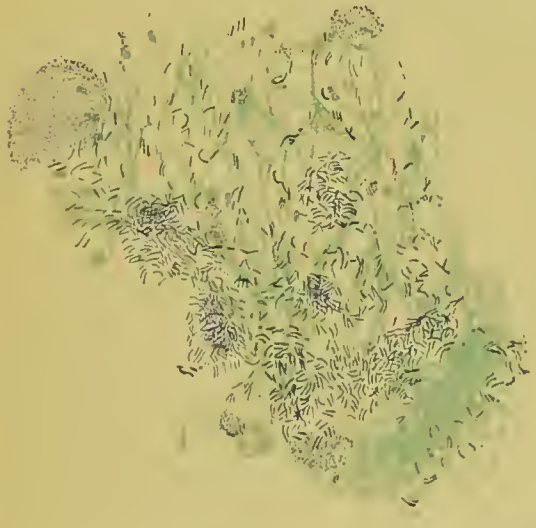


FIG. 96.—Diphtheria bacilli, from membranes taken during life. Staining with Löffler's potassium-methylene-blue, washed with water.

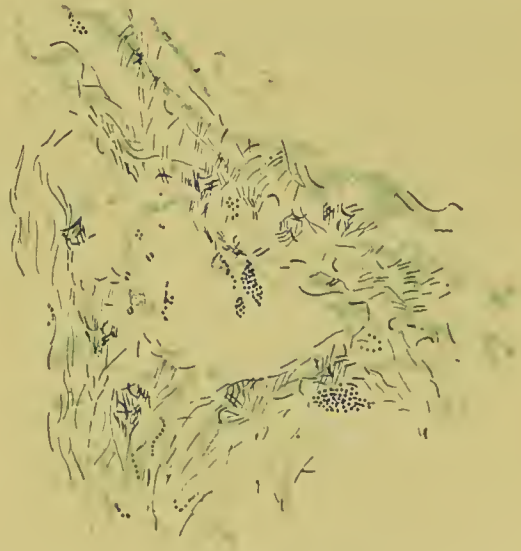


FIG. 97.—Diphtheria bacilli with mixed cocci. Preparations from membrane taken during life. Staining with Löffler's potassium-methylene-blue, washed with water.

important in order that specific treatment may be instituted as early as possible.

The loss of time by the plate method is, as was mentioned above, at least twenty-four hours. In a doubtful case, therefore, it is best to use the specific treatment before the final decision is reached.

[1] See Appendix.

[2] See Appendix.

Mode of Procedure.—A small piece of the membrane or of a plug from a lacunar tonsil is to be removed with sterilized pincers and teased on the cover-glass with a sterile microscope-needle; then it is to be pressed carefully (not too much) between this and a second cover-glass. These are next to be separated one from the other, dried, and stained with Löffler's potassium-methylene-blue solution (30 c.c. of alcoholic solution of methylene-blue; 100 c.c. of 0.01 per cent. liquor potassii); rinsed in water.



FIG. 98.—Diphtheria bacilli (Löffler) from bouillon culture. Stained with Löffler's methylene-blue—Zeiss homog. immers. $\frac{1}{12}$, oc. 4.

Mode of Procedure for a Bacteriological Diagnosis of Diphtheria.—An extended series of parallel experiments has shown us that for a quick and reliable demonstration of the presence of diphtheria bacilli it is by far the most advantageous to remove a particle of the exudation or of a plug. We have therefore abandoned the different methods



FIG. 99.—Spoon-forceps (natural size).

of wiping or scraping off of the exudation with pledgets of wadding, sponges, loops of platinum, or spatules, and always use a well-sterilized long, sharp, spoon-pincers, with which it is easy, with a little practice, during the inspection of the pharynx to obtain a small particle of exudation and even of a deep-seated plug. If it is desired both to use the microscope and to make a culture, it is best to remove two particles—the first from the pincers with a sterile needle, and to leave the second in the pincers till a culture is made. The pincers are sterilized every time immediately after use by boiling in a solution of soda, and preserved in a sterilized test-tube closed with a pledget of wadding. After use the pincers are immediately put into the test-tube again, together with the particles which have been removed, and left in the tube until the culture is made. The pincers may be carried in the pocket enclosed in a test-tube of strong glass.

The particles which are destined for the microscope are placed with sterile needles between two carefully cleaned, strong cover-glasses, which are to be pressed firmly together, then separated, dried, and stained with Löffler's potassium-methylene-blue, and rinsed in water. The particle destined for culture it is best to spread in three lines upon congealed Deicke-agar plates.

In a case of suspicious croupous laryngitis without disease of the pharynx, the pharynx, if possible, is to be wiped energetically down to the epiglottis with a sterile pledget of wadding to be used for the cul-

ture. The microscopical preparation from such a case is generally without diagnostic result. In examining a case of convalescence from diphtheria for possible still present bacilli, the tonsils are wiped and the culture made in the way just described. A like method is pursued if there is an exceptionally deep-seated, suspicious tonsillar plug.

The culture of diphtheria bacilli and its results are discussed in the Appendix.

A luxuriant *growth of leptothrix in the pharynx* (*pharyngomycosis leptothricia; algosis faucium leptothricia*) may occur in the form of long-existing, quite or almost quite unirritating lacunar plugs, which, on inspection of the tonsils, either look like common plugs or appear flatly imbedded in the mucosa, and are then scarcely perceptible.

In individual cases this affection may extend from the tonsils particularly to the follicular glands at the root of the tongue, or even still farther into the trachea itself. They manifest themselves as a number of distinct, elevated, yellowish-white specks. The nature of these deposits is easily recognized under the microscope, especially after the addition of iodin[1] (compare further regarding leptothrix buccalis, page 257).

EXAMINATION OF THE ESOPHAGUS.

Preliminary Anatomical Remarks.—The esophagus begins at the level of the cricoid cartilage of the larynx (= the lower border of the sixth cervical vertebra), and extends to the stomach at about the height of the base of the xiphoid process. At first it lies immediately in front of the vertebræ; then it comes a little forward, and at about the seventh dorsal vertebra it bends a little to the right, then again to the left, to reach the esophageal opening in the diaphragm. In adults the esophagus is about 25 cm. long. When we employ an esophageal sound[2] we estimate the distance from the incisor teeth to the stomach at about 17 cm. in the newly-born, while with adults it is about 40 cm. In the latter the distance from the incisor teeth to the bifurcation of the trachea is about 22 cm. The esophagus does not have the same diameter throughout: its narrowest points are at the commencement and where it perforates the diaphragm. The neighboring organs with which it has important relations in different diseases are—the trachea for the upper 7 to 8 cm. of the esophagus, the bronchial glands, the pleura, the pericardium, the aorta from the bifurcation of the trachea downward; lastly, the recurrent nerve from the bifurcation upward. It is only in the neck that the esophagus can be felt from without. Below the neck we cannot employ the usual methods of examination.

Characteristic distress almost always occurs with certain diseases of this organ—namely, with those conditions which result in *stenosis* (stricture): there is more or less deeply-seated difficulty in swallowing; the patient after taking food has a feeling of pressure, or even of pain, in the neck or the chest—a feeling that what has been taken cannot be passed down. According to the place or degree of the

[1] See p. 257.

[2] See next page.

stenosis, the patient experiences difficulty only after taking large, slightly comminuted bites of food, or even after swallowing soup or fluids, either immediately after the former or only after many bites or swallows. Moreover, the food may be regurgitated, wholly or in part, some time after it has been taken. Then we distinguish it from vomiting by the absence of odor, of acid reaction, and of muriatic acid. Pain in swallowing, without stenosis, occurs with inflammation of the mucous membrane of the esophagus or in its near neighborhood (mediastinum).

Examination of the esophagus itself is almost confined to direct palpation from within by means of the sound, excepting that in the cervical portion we can employ inspection and palpation from without. Auscultation furnishes little aid; the same is true of percussion. But it is very important in many cases to examine the neighborhood, particularly the thorax.

Only in exceptional cases do *inspection and palpation of the cervical portion* yield any result, because the great majority of diseases of the esophagus are located quite below the bifurcation of the trachea. We can feel a *carcinoma* of the cervical portion (likewise swelling of the glands of the neck); we can feel, and often also see, pulsating diverticula when they are full—that is, after the patient has eaten. Carcinoma of the lower end of the esophagus can be felt from the abdomen if the cardiac end of the stomach is encroached upon. *Pain* from pressure in the neck occurs in the conditions above named and in inflammations, as, for instance, after swallowing acids and alkalies.

Direct Palpation; Examination with the Sound.—For diagnostic sounding of the esophagus we employ either a whalebone or a French or English (black, yellow, or red) hollow and fenestrated esophageal sound. We think the solid, so-called esophageal bougies can be dispensed with. The whalebone sound consists of a thin staff with an olive-shaped ivory knob screwed upon one end. We have knobs of different sizes in order to determine and measure the degree of the stenosis.[1] Before using we are to make certain that the bulb is secure upon the staff, and also that the whalebone is perfectly smooth, so as not to catch anywhere or to cause injury. This sound furnishes the most positive information, and yet its use requires the greatest dexterity and caution. The cylindrical India-rubber sounds (especially the English esophageal sounds, which are the hardest) before using must be somewhat softened by dipping in warm water. Begin the examination with a thoroughly softened rubber sound, and only resort to the whalebone later.

Before introducing it we are to moisten the rubber sound its entire length, or the knob of the whalebone sound, with white of egg, glycerin, or with olive oil (but not with water). The patient, with the chin somewhat elevated, sits upon a chair or the edge of the bed. The index and middle fingers of the left hand are introduced into the mouth, and with them we slowly feel as far as the root of the tongue. Then we seize the sound with the right hand, like a pen-holder, and slowly push it along the tongue under the two fingers. As soon as the point of the sound passes beyond the ends of the fingers we press its end

[1] See below.

somewhat downward with the tips of the fingers, and at the same time elevate the right hand, so that the sound may not strike against the back of the throat. The sound is then with gentle pressure pushed on, always holding it as if writing. The left hand is now withdrawn.

Special precautionary measures, such as placing a cork between the teeth or anything to hold the jaw, are usually not necessary, since this operation is not performed upon unwilling or unconscious patients (it is otherwise when sounding the stomach).[1] Only with children are we sometimes obliged to use the cork. Many patients bear a skilfully-performed sounding very well, but others can only become accustomed to it from considering its beneficial results. If the motions of strangling are not severe, we need not be disturbed by them, but if there is vomiting, we must at once withdraw the sound in order that there may be no choking. A slight spasm of the glottis and momentary arrest of breathing have no significance, yet attention is called to the second paragraph below. On the other hand, it is disagreeable if the point of stenosis bleeds during the sounding. The procedure can only be repeated with care after an interval of several days. In most cases of this kind the sounding must be abandoned.

We sometimes meet with *a resistance* which is *not pathological:* 1. At the posterior wall of the throat, but only with unskilful introduction of the sound.[2] 2. Sometimes, if the cricoid cartilage of the larynx somewhat overlaps the esophagus, the point of the sound strikes against it. This is easily passed by withdrawing it a little and then pushing it on again. 3. By spasm of the esophagus caused by the sound, which disappears soon by waiting.

The *life* of a patient may be *endangered* by several occurrences: *The introduction of the sound into the trachea* happens very rarely. At any rate, as soon as there is marked difficulty in breathing the sound is to be withdrawn. If the patient is able to pronounce *ah* clearly, or if the portion of the sound introduced is longer than the trachea, then we know that it has not entered the trachea. Other signs are deceptive. A still greater danger is that *the wall of the esophagus may be injured or ruptured.* This results from narrowing of the canal if it has become thin and fragile from a crumbling new formation, or by an ulceration, or when an abscess or aneurysm near the esophagus is thus perforated. The results of these are either ichorous mediastinitis or pleurisy with fatal termination, or, if an aneurysm, with immediately fatal hemorrhage. *We must never employ force if the sound meets with resistance.* If there is the suspicion of an aneurysm founded on an examination of the chest, of the blood-vessels, etc., under all circumstances we are to omit using the sound.

Examination with the sound gives information in the following ways:

1. Sometimes a *deep-seated pain* occurs after the examination has been made several times, although the sound has only been introduced a certain distance and it has not met with any resistance. It may depend upon inflammation in that neighborhood,[3] upon an ulcer, a carcinoma not causing stenosis, a purulent esophagitis, or periesophagitis.

[1] See Sounding the Stomach.

[2] See above.

[3] For determining its height, see under Stenosis.

2. The sound meets with *resistance*. Then the patient in many cases is sensible of pressure or has a sensation of pain; sometimes there is severe strangulation. We move the sound back and forth, and endeavor to advance it with very slight pressure. We mount a smaller knob upon the whalebone sound or take a thinner rubber one. But the smaller the sound the greater the danger, and hence greater caution is required in using it.

If we are at length able to advance it farther, then we feel resistance just so long as the knob is in the stenosed portion. After passing the narrowed part it again passes easily, but of course meets with resistance at the same point as it is withdrawn. In using the rubber sound we certainly feel the resistance becoming somewhat less as the stenosis is overcome, but in any case the resistance continues so long as the sound is in the stenosed part.

We obtain information: (*a*) Regarding the *situation* of a stricture by bearing in mind the rules given when referring to the anatomy of the parts. We introduce the sound as far as the stenosis, note the location, starting from the incisor teeth (by seizing the sound accurately with the fingers), draw it out and measure it.

(*b*) Regarding the *degree* and *length* of the stenosis: we learn the former by the thickness of the sound that will just pass the stricture; the *length* of the stricture will best be ascertained by employing whalebone sounds, in that we can mark the place where the incisor teeth touch the sound when it enters the stenosis and as it passes through the stenosis, and then measure the difference. Also, if there is a double stenosis, it is indicated (see Fig. 100).

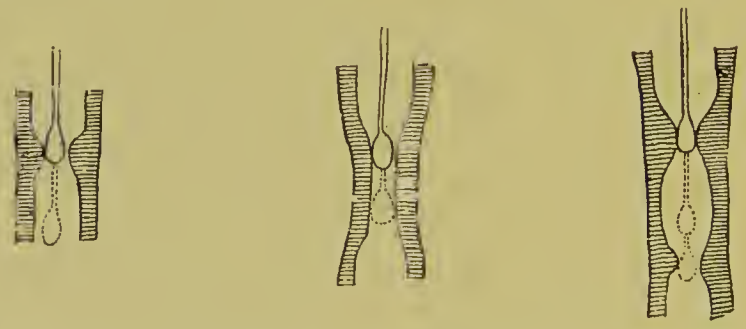


FIG. 100.—Diagrammatic representation of sounding the esophagus when there is a short, a long, and a double stenosis.

We can learn almost nothing regarding *the nature of the stenosis* unless we should catch in the fenestrum of an India-rubber sound a shred of tissue which would enable us to diagnose a carcinoma, or unless we should meet with the condition described in the next section (3).

3. By repeated introduction of the sound we are sometimes able to pass it through, but if again we meet with an insuperable obstruction, we must be very careful: this points to a diverticulum, though not indeed with absolute certainty, since it may be met with in other kinds of stenosis.

4. In a case of stenosis which we have repeatedly examined we

suddenly find ourselves unable to get the smallest sound through where it has frequently passed easily. This may indicate an *obstruction* by a foreign body, as was the case in one instance under my observation which ended fatally, where a cherry-stone was found in the stenosis.

5. Sometimes the sound has an unusually extensive lateral movement above the stenosis. This indicates a considerable dilatation of the esophagus above the point of contraction. Such dilatations particularly develop in severe and long-existing stenoses.

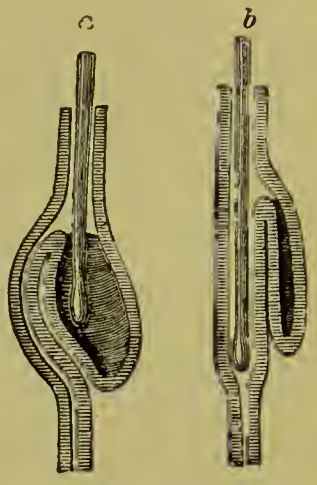


FIG. 101.—*a*, sounding the esophagus when the diverticulum is full; *b*, sounding when the diverticulum is empty.

Stenoses at the cardia are the consequence either of cicatrization or carcinoma. For a differential diagnosis we take into consideration, first of all, the age of the patient and former anamnesis (swallowing some corroding liquid, particularly lye). As regards the present condition, the presence of particles of carcinoma in the eye of the sound, or of a tumor perhaps in the epigastrium on the left anterior wall of the stomach, decides the case as one of carcinoma; but both of these conditions are rare.

Stenoses above the cardia, in the lower part of the esophagus, are generally carcinomatous. If they appear after the fortieth year of life, they are almost always of this nature. Nevertheless, of course even here we must not overlook a possible finding of particles of tissue [which can be examined with the microscope].

Farther above, in addition to carcinoma, the diverticula due to internal pressure [*Pulsionsdivertikel*] come into consideration. Their peculiar signs when examined with the sound have been mentioned above. The diagnosis becomes comparatively easy in the part of the esophagus which lies in the neck. Here a tumor with greatly varying volume indicates a diverticulum due to internal pressure.

Examination of the neighborhood of the esophagus—that is, of the neck and thorax—is of the greatest importance. We are thus able to discover compressing tumors or to exclude them with probability. We may aid the diagnosis by giving attention to the *larynx* and observing whether there is a recurrent paralysis, which may exist even though the voice be quite clear. Compression of the recurrent nerve sometimes occurs in carcinoma of the esophagus, with aneurysm of the aorta (particularly the left nerve). Moreover, we take into consideration the examination of the chest, especially whenever there is any evidence of a rupture, as in pleuritis, gangrene of the lungs, rupture into the trachea or bronchus, with coughing up of particles of food; pericarditis and emphysema of the skin.[1]

Percussion of the esophagus itself can be of almost no aid. Large diverticula in the neck may show dulness, provided they are full. Exceptionally, a dilatation above a stenosis of the cardia may be discovered, if dilated with food, by dulness at the back and to the left,

[1] See Examination of Skin.

or more rarely to the right, of the spinal column from the lower boundary of the lung toward the middle of the scapula.

Auscultation of the esophagus has no independent diagnostic value.

Swallowing.—Normally, the morsels of food or the liquid to be swallowed, after reaching the root of the tongue, are pressed or squirted very energetically into the esophagus by the contractions of the mylohyoidei, hyoglossi, and of the constrictor pharyngis, while the palate and tongue close the exit upward. The morsels pass through the esophagus itself without difficulty, whilst the passage of larger morsels through the cardia is probably assisted by the muscles of the esophagus. Corresponding with the moment of swallowing, there is produced a primary noise, and about five seconds later, during the passage through the cardia, a second noise. The first sound has been called by the not very elegant name of *squirting noise*, the second the *pressing noise*.

The first sound is heard most distinctly at the pit of the stomach to the left of the xiphoid process, and also at the same level on the back to the right and left of the spinal column. It is heard also farther upward, particularly on the back, but here it often becomes an uncharacteristic clucking. This noise is without any diagnostic value, because in health it is unequally distinct, and it may also be absent.

The pressing noise [the second sound], however, is heard rather constantly in health at the above-mentioned places, which correspond with the cardia. But it is absent if there exists a considerable stenosis at the cardia or above it.

Esophagoscopy has hitherto not given results which compensate for the considerable inconveniences for the patient which are connected with the different methods at present in use. We therefore think it is better to omit it for the present.

EXAMINATION OF THE STOMACH.

Topography of the Abdomen.—This is represented in the accompanying figure. We form the different sections by prolonging the mammillary lines (or a line which passes from the middle of Poupart's ligament upon each side); also by lines which, in the upright position, are drawn through the ends of the eleventh ribs and through the anterior superior spines of the ilii. By these latter lines the section lying between the mammillary lines is divided into the *epigastrium*, *mesogastrium*, and *hypogastrium*. It is further to be added that the region directly over Poupart's ligament, which extends inward toward the symphysis pubis and outward somewhat over the middle of the ligament, is called the *inguinal region*, and the territory below the ends of the ribs the *hypochondrium*. So far as the abdominal contents are parietal, their relations to the separate regions of the abdomen are plainly indicated in the accompanying figure.

Anatomy of the Stomach.—Only a little more than the pyloric portion [one-sixth] of the stomach lies in the right half of the body, the rest [five-sixths] being on the left of the median line. It slopes obliquely from the left downward toward the right, so that the *cardia* is about behind the sternal insertion of the seventh rib, *the pylorus* between the right sternal and parasternal lines, on a level with the apex of the

xiphoid cartilage. *The fundus*—the portion situated the highest, clinging to the left side of the dome of the diaphragm—rises as high as the fourth intercostal space. *The lesser curvature* forms a bow with its

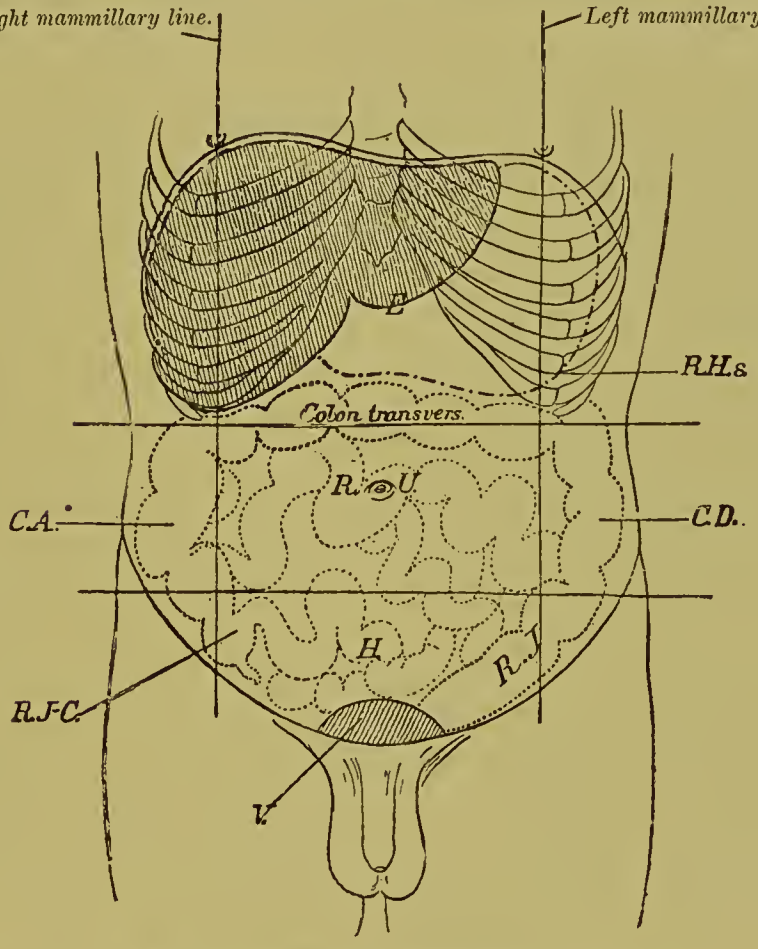


FIG. 102.—Position of the abdominal contents.

CA, ascending colon; *CD*, descending colon; *RJ-C*, ileo-cecal region; *RJ*, inguinal region; *RHs*, left hypochondrium; *EE*, epigastrium; *RU*, umbilical region; *H*, hypogastrium; *V*, bladder.

convexity disposed obliquely downward toward the left. It, with the cardia and pylorus, which it connects, lies more posteriorly, covered by the liver, while *the greater curvature* extends forward toward the abdominal wall, so that a line drawn from the lowest point of the lesser to the lowest point of the greater curvature would incline forward and downward. The situation of the greater curvature varies very much with the degree of distention of the stomach. In health, however, it only very exceptionally extends to the umbilicus.

The fundus of the stomach is adjacent to the diaphragm, the spleen, and the left kidney; its greater curvature and also the lower part of its posterior surface to the transverse colon; the pylorus, lesser curvature, and that portion of its anterior surface which is near to these to the left lobe of the liver. Behind and above the stomach, situated at the upper part of its posterior surface, is the sinus of the peritoneal cavity, the bursa omentalis (pathologically not unimportant), and also the pancreas.

When the stomach is moderately distended a part of the anterior surface and the greater curvature are parietal, so far as they are not prevented by the lung or heart from above, or by the spleen on the left, and by the left lobe of the liver on the right. That part of the parietal surface of the stomach which is covered by the left lower portion of the ribs comprises the important region to which Traube gave the name of "*halfmoon-shaped space.*" We see from this description that with moderate distention only a small part of the healthy stomach can be directly examined. The most important parts, the cardia and pylorus, are bent deeply in. But we have a favorable moment for examining the latter in certain pathological conditions, where it is desirable to be able to judge of it, it being often pushed down with the lesser curvature below the liver.

Inspection and Palpation of the Stomach.—There is scarcely any place where inspection and palpation are so closely connected as at the abdomen, and especially the stomach. The patient is placed so as to lie comfortably, with the upper portion of the body moderately raised. We look at the region of the stomach with the greatest care, illuminating it from all possible directions: then palpate with the tips of the first, second, and third fingers, and thus notice first the tenderness (always at first proceeding very cautiously), then the objective condition; finally completing the palpation with inspection, or *vice versâ*.

The result of the two methods of examination will be affected by several factors—by the size, sharpness of the boundaries, and density (resistance) which we discover in the abdominal wall, and its condition. As regards the latter, it is important for the examiner to avoid causing contraction of the abdominal muscles by having the patient in the recumbent posture, cautioning him to keep the muscles lax, and by proceeding slowly with the palpation, the hands being warmed. Contraction of the recti abdominales, with their short tumor-like sections of muscle, may very much disturb, or even deceive, one in making an examination. As to the general thickness of the abdominal walls in chronic diseases of the stomach, especially if very severe, this is very much lessened by wasting—a condition favorable for making an examination.

The normal stomach cannot at all distinctly be recognized or defined through the abdominal wall. It can only exceptionally be done when there is extreme emaciation.

Not infrequently there are cases where, in extremely wasted females with very lax walls, the greater curvature and peristalsis of the anterior wall of the stomach could be clearly seen. In these cases the autopsy shows a normal condition of the stomach.

On the other hand, *the healthy stomach*, distended with food or gas, sometimes enables us to imagine its condition by the projection in the epigastrium, and still more by a high halfmoon-shaped space—that is, by tympanitic resonance over the left lower lobe of the lung in the side.[1] We can sharply bound a healthy stomach only in individual cases when it is inflated with gas.[2] Thus, it has been found that the greater curvature of a normal stomach, when very greatly distended,

[1] See under Percussion.

[2] See Method of Procedure, p. 271.

may reach as far as the umbilicus. Of course we cannot ascertain the location of the lesser curvature if the stomach is in its normal position. Moreover, the distensibility of the healthy stomach varies very much with different persons, so that on trial one person earlier, and another later, has difficulty, especially oppression, which marks the limit of distention.

The *chief pathological signs* furnished by the stomach are: its distention or displacement, its thickness, and amount of peristaltic action of its walls, also signs of circumscribed tumors in its walls. Other important signs are to be added to those already mentioned. Pain upon pressure during palpation requires a special description.

Distention is more or less distinctly made out by inspection and palpation, according to its extent and the thinness of the abdominal walls. But it may also entirely elude examination. In favorable cases we can see and feel (easily when looking down from the patient's head) the greater curvature. To a varying extent it moves down, often below the umbilicus, more rarely nearly to the symphysis, and in so doing it shows the bend toward the left. The position of the greater curvature of course varies with the degree of fulness of the stomach, but usually, unless artificially emptied,[1] as by emesis or the stomach-pump, it does not come up above the umbilicus. At the same time the pyloric portion very often has a peculiar behavior which influences the whole situation of the stomach and renders the pylorus as well as the lesser curvature accessible for examination. When the stomach, for the time being, is distended by a large quantity of food, in the upright position of the patient it pulls the pylorus forward from under the liver, and with it, under some circumstances, the lesser curvature. This, in rare cases, is seen in the upper epigastrium, in a line convex downward (when the light falls from the foot of the bed), when sometimes it may even be felt. Also the *portio pylorica* and even the normal pylorus may be felt.[2] In consequence of this displacement of the pylorus the whole stomach slopes more strongly downward toward the right.

In rare cases the pylorus has this low down position, without there being any dilatation of the stomach. The condition is congenital or caused by strong adhesions (Kussmaul).

As has already been mentioned, the distinctness with which the figure of the stomach can be made out is largely influenced by the extent of its fulness. Hence, for the purpose of making the examination we must *artificially distend it* (Frerichs). Until very recently this was always done with carbonic acid, by giving the patient as much as two teaspoonfuls of tartaric acid and bicarbonate of soda dissolved in a little water. The gas quickly develops in the stomach, and demonstrates clearly the situation and size of the organ, rendering the examination of its walls easy.[3] But this procedure sometimes gives rise to a feeling of oppression, and even of symptoms of collapse. Recently there has been devised a method of inflating the stomach which is much more to be recommended, because the amount of gas for distending the stomach can be regulated exactly, and, if necessary, it can be emptied out in an instant. A soft stomach-tube is introduced (just as in sound-

[1] See under Contents of the Stomach.

[2] See under Tumors.

[3] See under Peristalsis and Hypertrophy.

ing the esophagus), and then the stomach is inflated with air through the tube by means of an India-rubber ball, introducing as much as is necessary or as the patient can bear. At any time the air can immediately be let out through the tube.

By inflating the stomach the so-called *hour-glass stomach* can be easily recognized during life (twice it was formed by a scar which strictured it in the middle). In the same way we can discover that the pylorus does not close, by the fact that the gas blown in does not distend the stomach, but immediately enters the small intestine.

Von Ziemssen still gives the preference to distention with carbonic acid—a method which we will not omit to mention. In his last communication he gives the proportions for adult men as 7 grams of bicarbonate of soda and 6 grams of tartaric acid; for adult women 1 gram less of each.

The sound may be employed in the same way as with the esophagus to determine stenosis at the cardia due to cancer. (The employment of a hard English esophageal sound for ascertaining the size of the stomach [Leube] is scarcely to be recommended. The sound is introduced into the stomach, and pushed on until it meets resistance at the greater curvature, and then we ascertain where the end of the sound is by palpation from without.)

Regarding *palpation by striking* and the resulting *splashing*, see under Auscultation. In the neighborhood of the stomach we may have epigastric pulsation,[1] liver-pulse;[2] lastly, it may be communicated from the aorta or from aneurysm of the abdominal aorta. With tumors of the stomach the pulsation from the aorta is usually very distinctly transmitted.

Increased resistance; peristaltic motions. The former occurs simultaneously with the general distention of the stomach in consequence of the hypertrophy of the muscular portion which generally accompanies dilatation of the stomach. Hence it is an indirect sign of dilatation. If it is found within a limited area, as in the right half of the epigastrium, even if it is not sharply defined it may indicate carcinoma. We must be careful not to confound it with contraction of one of the bellies of the rectus abdominis. *Peristaltic motions* which can be felt as well as seen are very important, being often the first signs of an hypertrophy, and thus a dilatation. By their situation and extent they may also indicate the size of the stomach. It is very rare for them to occur without dilatation: only in nervous "peristaltic unrest" of the stomach (Kussmaul). Generally they extend in the normal direction from the fundus to the pyloric region. But sometimes, and that in marked pyloric stenosis, they are reversed—*antiperistalsis*. They will often be excited or increased by gentle strokes and by faradization; sometimes by irritation of the skin, as by simply uncovering it. With very lean persons we must think of the possibility of there being, under some conditions, intestinal peristalsis.

Tumors in the region of the stomach are often only to be felt, not seen. They cannot be demonstrated if connected with a part of the stomach that is not parietal—cardia, lesser curvature, posterior wall of the stomach, commencing cancer of the pylorus. These tumors are

[1] See p. 177.

[2] See pp. 221 and 228.

most frequently cancer of the stomach (more rarely a dense scar from ulcer), and are most often located to the right of the middle line, because they belong to the portio pylorica or to the pylorus itself. In the latter case they can generally only be felt when the pylorus is pushed downward, as has already been mentioned. Carcinoma usually feels uneven and dense. Less frequently it is smooth, and can then easily be overlooked or be mistaken for a belly of the rectus.[1] Projection of the stomach during deep breathing, as a result of the movements of the diaphragm, usually does not take place at all, for the reason that the stomach is not a solid body. We observe a slight, or possibly a marked, *respiratory displacement* when there is adhesion of the distended pylorus and the liver,[2] or if there is a tumor which extends from the subphrenic region to a parietal portion of the stomach. Dense scars from ulcers and the infrequent hypertrophy of the pylorus, also solid bodies that have been swallowed, may feel like tumors. Mistaking them for scybala in the transverse colon[3] is not likely to happen.

In all diseases of the stomach *tenderness* during palpation may be wanting. It is absent least frequently with ulcer of the stomach. If there is pain, it may vary very much: in acute catarrh of the stomach, also sometimes in chronic, it is dull and quite diffuse; with ulcer it is often very much circumscribed, limited to a spot the size of a dime, extremely severe, often shooting through to the back, especially toward the left; in carcinoma there is sometimes a marked insensibility, sometimes a more diffuse, sometimes a narrowly-defined, pain of variable intensity.

A constant circumscribed tenderness in the gastric region and severe spontaneous pain, which are markedly increased by movements of the body, according to the observations of Landerer, may also be produced by adhesions of the stomach or omentum to the abdominal wall. The cause of the adhesion in such cases is most probably a former ulcus ventriculi or a former circumscribed trauma. Such cases are very difficult to distinguish from nervous cardialgia or splanchnic neuralgia.

Percussion of the Stomach.—This applies to that portion of the anterior wall of the stomach which lies against the abdomen and the anterior (left lower) wall of the thorax. It yields, in much the greater majority of cases, a very deep tympanitic sound, and sometimes, when there is marked tension of the stomach, a clear non-tympanitic sound. If the stomach contains a considerable amount of food, it may, in part (especially in standing), have an absolutely dull sound. But we hardly ever find it dull throughout the whole extent of that portion of the stomach that is parietal, because it almost always contains considerable gas as well as food. The tympanitic as well as the non-tympanitic stomach-sound frequently has a *metallic quality*.

The boundaries of the stomach are determined by topographical percussion (see Fig. 103). They are as follows:

On the side toward the liver there is a dull sound; it is often difficult to make out because the border of the liver is thin.[4] On the side toward the lung there is a non-tympanitic, clear sound. Here it is

[1] See under Resistance. [2] See this. [3] See Intestine.
[4] See Percussion of the Liver.

often difficult to mark sharply the boundary-line, on account of the thinness of the border of the lung and the similarity of the two sounds.

Sometimes we have to distinguish a boundary of the stomach from the heart, should the apex of the latter reach farther toward the left than the liver; sometimes from the spleen if the stomach should be stretched out somewhat. We can separate it from the large and small intestines, both of which give a tympanitic sound.

Except these last named the boundary-lines are all dependent upon the situation and size of the surrounding organs. Therefore, and because there are no true boundary-lines for the stomach, except its parietal boundaries, we do not employ percussion for the stomach.

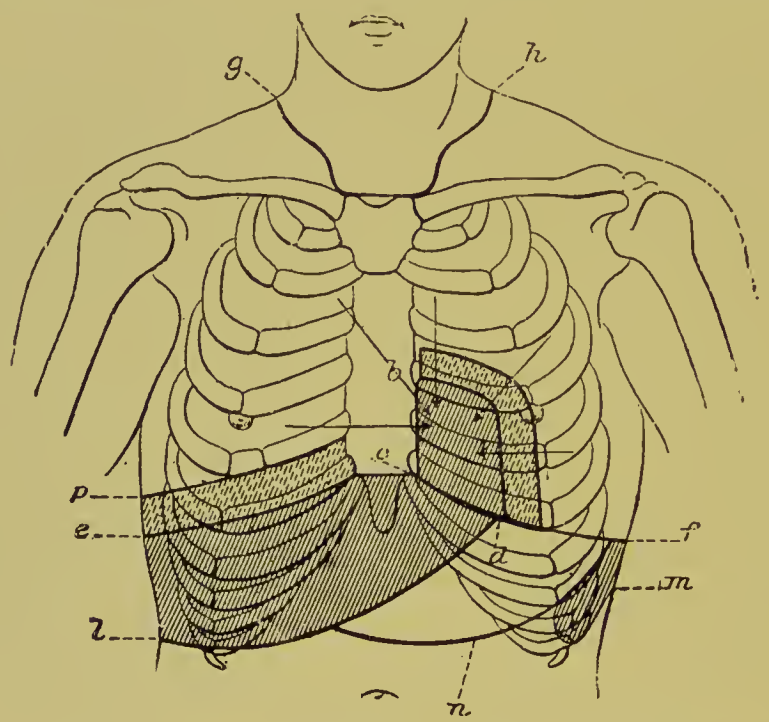


FIG. 103.—Percussion boundary of the lungs in front (Weil).

g, *h*, the upper boundary of the lungs; *e*, *f*, the lower boundary of the lungs; *b*, *d*, boundary between the lung and heart at the incisura cardiaca. The darkly-hatched surface represents the portions of the heart and liver that are in contact with the chest-wall; the light hatching, the so-called relative heart- and liver-deadness (see later). *m*, spleen-deadness; *n*, the average position of the lower boundary of the stomach.

The only real boundary is that on the side toward the intestine, which gives the situation of the greater curvature.

But it is almost always very difficult to determine this line (there being a tympanitic sound on both sides of it, with only a difference in pitch). We can hardly even maintain its correctness without the aid of inspection and palpation. Thus, percussion of the stomach, for the great majority of cases, has an extremely doubtful value.

On the whole, we get the best results from percussion in health, and particularly when the stomach has been *artificially dilated*. With the former we then find that the greater curvature usually is somewhat above the umbilicus, sometimes reaching beyond it. When the stomach is moderately full it commonly stands below the umbilicus, between the apex of the xiphoid process and the umbilicus. If the stomach is

dilated, the boundary is lower down.[1] Likewise, should the lesser curvature be lower down, it can be made out by the aid of percussion.

Another procedure, but one which is not always successful, is first to empty the stomach as much as possible,[2] then to percuss the abdomen, the patient being in the standing position. Usually we do not find any boundary for the stomach. Then we have the patient drink freely, and again percuss while he is standing. In the lower part of the stomach, hence above the greater curvature, about in the middle line, we shall find a dulness which indicates the situation of the greater curvature, and thus a possible dilatation may be recognized (modified after Penzoldt). This dulness may sometimes be directly proved, without any preliminary procedure, if the stomach is partly filled with fluid. The dulness disappears when the patient lies down.

There is distinct dulness with *tumors of the stomach* (strong percussion) only when they are very thick, and this is not often the case. Hence they usually give stomach-resonance. But tumors of the liver and spleen, on the other hand, almost always are dull because they are larger. Yet this difference is not an entirely sure sign.

Rod-pleximeter percussion[3] over the stomach usually gives a beautiful silver tone. It is employed for determining the boundary under the supposition that in this way the person who is listening over the stomach must hear the high silver tone just so long as his assistant percusses over the stomach; but the result of this procedure is hardly ever positive enough to give it value.

That part of the left lower lobe of the lung is designated as the "*circular stomach-lung space*," where a tympanitic sound may be heard with strong percussion (Ferber). We may likewise speak of a "circular stomach-liver space," sometimes even of a "stomach-heart space."[4] None of these have any value for exactly determining the size of the stomach.

The Halfmoon-shaped Space (Traube).—This is the name given to that portion of the lower left part of the thorax which lies below the lung (or heart), between the liver and spleen, and, as a rule, in health gives a tympanitic sound, most frequently a stomach-sound, but not infrequently also an intestinal sound, or both. It is discovered by gentle percussion. Occasionally, in health, we here find dulness instead of tympanites, and then only when the stomach is decidedly full, or when the full transverse colon is here parietal, or when the greater omentum is unusually loaded with fat (Weil).

In *enlargement of the liver, of the left heart*, or *of the spleen* this space will always be found correspondingly smaller. But its behavior in certain conditions of the left lung or of the left pleura is of especial diagnostic interest. *Exudation in the left pleura* usually causes dulness correspondingly early in the upper portion of this space in that it first collects in the complementary pleural sinus. As the exudation increases, the halfmoon-shaped space diminishes more and more, the dulness sometimes extending as far as the bend of the ribs, depending upon the amount of downward pressure of the diaphragm, unless there are pleuritic adhesions in the pleural sinus, in which case

[1] See Inspection, Palpation. [2] See Emesis. [3] See p. 117. [4] See p. 179.

we do not have the space diminished. As the pleuritic exudation is absorbed the space resumes its normal proportions, and if there is shrinking after the absorption, it becomes greater than normal, for the reason that the lower border of the lungs does not again come down to its former place, and, on the other hand, the diaphragm stands higher. Rarely, with *pneumonia* of the whole left lung or its lower lobe the halfmoon-shaped space becomes very slightly smaller as a result of the enlargement of the lung during hepatization, and also, probably, from a small pleuritic exudation.

It is to be observed that in acute disease of the left half of the chest an early distinct diminution of the halfmoon-shaped space is made manifest by a certain degree of dulness; a marked diminution of the space indicates, almost to a certainty, a pleuritic exudation; and if there is extensive dulness in the left half of the chest, if the differential diagnosis between pneumonia and pleurisy is uncertain, then a decided diminution in the size of the space speaks with strong emphasis in favor of the latter.

Auscultation of the Stomach.—This has value in only one direction, but that is not to be undervalued. When *palpation* is made *by strokes* upon the region of the stomach, striking more or less strongly, according to the sensibility of the patient, very short blows with the tips of the fingers, sometimes spontaneously, and again only with the strokes, we hear a splashing which is loud enough to be heard at a distance. This results from a certain relation between the fluid and the gas in the stomach even in health, but very much more frequently in dilatation. Hence in making a careful examination of the stomach we must always employ it. In itself it does not indicate anything, even though it is often found when the examination is frequently repeated.

Such a splashing sound or a similar one may also have its origin in the intestines and even in the peritoneal cavity. As an intestinal sound it is heard in profuse diarrhea, most markedly in cholera; also sometimes in intestinal occlusion. From the peritoneal cavity a somewhat similar sound may be heard in circumscribed perforating peritonitis, particularly in subphrenic abscess.[1]

If we apply the ear when the stomach is inflated with carbonic acid, we shall hear a loud seething. We can recognize the same thing, but less distinctly, in dilatation of the stomach with fermentation of its contents.

Illumination of the Stomach; Gastro-diaphanoscopy.—The first method applicable in man by which an illuminating body, visible through the abdominal wall, could be introduced into the stomach, was devised by Einhorn in 1889. Einhorn's instrument is a stomach-tube which carries on its lower end an incandescent lamp surrounded by a glass shade. The conducting wires go through the interior of the hollow sound. This illuminating sound can be introduced into the stomach like any other stomach-sound, and the illumination effected by turning on the current without injury to the stomach, whether it be full or empty, for the amount of heat is very slight. The diagnostic results of diaphanoscopy are greatly heightened if a considerable quantity of

[1] See this.

water (up to 1500 c.c.) is introduced before or during the examination This may be done with the illuminating sound itself if an aperture is made in it directly above the lamp (Kuttner).

The illumination may bring into view a part of the greater curvature, and in gastroptosis also a part of the lesser, and also tumors located in the anterior wall may be perceived as dark places in the illuminated region. An important result of the method seems to be that the normal as well as the dilated stomach appears considerably larger if filled with water than has been hitherto supposed. Sources of error, as the illumination of neighboring intestinal coils filled with water and gas, it must be borne in mind, are not excluded.

I have no personal experience with the method, and therefore respecting the details refer to the writings of Kuttner[1] and Meltzing.[2] Whether diaphanoscopy really gives considerably better results than a carefully made examination of the stomach by inflation and percussion in a standing position certainly seems to me still doubtful, according to the results of these investigations.

Remark.—It is evident from the above that very often anatomical diseases of the stomach exist without any physical signs. Consequently, their differential diagnosis from nervous cardialgias and from some forms of nervous dyspepsia is frequently very difficult. In general, a certain uniformity of stomach complaints and their increase by moderate exercise of the body points to an anatomical disease. Most frequently, however, a positive differential diagnosis can be made by an investigation of the functions of the stomach. Therefore the examination of the *motive-power of the stomach during digestion and a chemical examination of its contents* frequently give much more important conclusions than the local examination. Therefore especial attention is called to the former.

EXAMINATION OF THE INTESTINES.

Inspection and Palpation.—In employing the former there must of course be illumination. The patient being in the dorsal position, we inspect the trunk, as a whole, from a distance; in detail, close at hand, palpating with a warm hand. Then, carefully grasping a part, we notice always first as to the amount of tenderness, when, if there is any suspicion of simulation or exaggeration, it is best not to ask whether we are causing pain, but simply to notice the result of moderate and also stronger pressure. After completing the first examination, which gives one the bearings of the case, inspection and palpation go, hand in hand, very closely together; for this reason we speak of them together.

Pain Produced by Pressure [*Tenderness*].—A diffuse dull pain often occurs with *intestinal catarrh.* A like diffuse, but generally an extremely severe, pain is observed with acute general *peritonitis.* Circumscribed tenderness is especially frequent in the right iliac fossa. It is often quite marked in *abdominal typhus* [typhoid fever], often more severe in *intestinal tuberculosis*, moderately severe in *typhlitis* and affections of the vermiform appendix, in both of the last-named diseases

[1] *Berliner klin. Wochensch.*, 1893. [2] *Zeitschr. f. klin. Med.*, Bd. 27.

generally (not always) in connection with other local signs.[1] Pain in the left iliac fossa is connected with the descending colon (especially *dysentery*). Very circumscribed severe pain at shifting points may occur with a circumscribed affection of the small intestine, as invagination[2] (intestinal tuberculosis). The seats of hernia require very especial attention. (Works upon surgery are to be consulted regarding these.) It is to be further remarked that pain in the abdomen, according to its location, may come from any of the organs contained in its cavity, and also from its walls; from the anterior abdominal wall (abscess); pain in the iliac regions from the hollow of the sacrum (inflammation, tumors); pains in the same place and in the lumbar region from psoas abscess.

The *dimensions of the abdomen* may be *increased:* by a layer of fat; by gas in the intestines (intestinal meteorism, tympanites), as it occurs continually, scarcely pathologically, after hearty eating, often with a large development of fat; but we may also have it in every variety of degree as a pathological condition: in acute and chronic catarrh of the intestine, intestinal stenosis, in acute and chronic peritonitis, and in abdominal typhus [typhoid fever], where it is often of diagnostic value. According to the amount of distention the abdomen is more or less full, which changes its normal soft condition to one of marked resistance. When there is marked meteorism the liver and diaphragm are pressed upon, and by the latter the lungs and heart are pressed upward.

In a case of typhus abdominalis [typhoid fever] I once saw an extensive inflammatory undermining of the abdominal wall, which very closely simulated meteorism by considerably distending the abdomen, which proved to be an abscess in the abdominal muscle. (For distention of the abdomen with fluid or air in the peritoneal sac, see Peritoneum.)

There may be *circumscribed distention of the abdomen* from a great variety of causes: most frequently from some condition in the peritoneum.[3] In chronically developing stenosis due to tumors or in acute incarceration it is produced in the intestines themselves; the piece of intestine immediately above the stenosis becomes distended. The correct diagnosis of such a circumscribed inflated piece of intestine is of great clinical significance, and must be sought for in every possible way. The chief point is to take time for inspection and likewise for palpation, observing carefully whether the flatulent portion is completely at rest or whether there is peristalsis,[4] whether the swelling changes its position in its entirety or not, or whether it is sometimes flatter or disappears altogether. Palpation and percussion give uncertain results; they may, however, particularly the former, sometimes serve to confirm what the eye has discovered.

Diminished volume of the abdomen (drawing-in, sinking-in) results from an insufficient amount of nourishment from any cause (especially from diseases of the esophagus, pyloric stenosis, any cachexia—in short, from any disease that requires (or results in) restricted diet. Usually this condition is more especially manifested by the absence of

[1] See below.
[2] See Palpation.
[3] Which see, and also the next page under Tumors.
[4] See this.

fat and wasting of the abdominal muscles. A particularly marked—the so-called "scaphoid drawing-in"—probably related to an active contraction of the abdominal muscles, occurs in meningitis, particularly basilar, and in lead-colic.

Intestinal peristalsis exceptionally can be seen when the abdominal wall is very thin and lax. It occurs almost exclusively in women who have had children (particularly if there is a separation of the recti muscles). On account of its similarity, it is to be distinguished from what is described below as pathological peristalsis only by the absence of other phenomena and by the narrowness of the intestinal figure.

Pathological peristalsis is an important visible and palpable sign of stenosis of the intestine, and occurs in the portion of intestine above the stenosis. We observe a round projection, with the slow motions of a worm, now disappearing and often immediately reappearing in a spot not far distant, so that we have the phenomenon of peristalsis. The intestine, as it becomes prominent, is moderately resistant and is often distinctly distended. [During the instant of greatest distention the prominence is more distinctly tympanitic.] The resistance may become greater in chronic stenosis of the intestine with hypertrophy. Sometimes the last swelling—that is, the one just above the point of stenosis—is the largest, and subsides with a loud cooing or bursting sound. This phenomenon may have a very great variety of manifestations, generally with a pressing, choking pain, and it may manifest itself under gentle blows, with faradization, or even by merely exposing the surface to the air. It is usually very difficult to draw any conclusion regarding the portion of the intestine involved by the location of the phenomenon or the direction of the peristalsis. On account of its thickness we are apt to mistake a dilated loop of small intestine for a portion of the colon.

Circumscribed tumors of the intestine are always felt before they can be seen. They may be—1. *Balls of feces, scybala*, in the large intestine, often recognized by being arranged in a circular form, by their location (which is often deceptive), or by their retaining an indentation. Sometimes we are only able to be positive regarding their nature by their disappearance after free purgation. 2. *Tumors of the intestine* are either new formations, which are generally very firm, uneven, or they result from invagination of one portion of the small intestine into another or into the large intestine, which form round vermiform tumors. The former are entirely fixed, the latter may suddenly disappear. Both may be connected with signs of stenosis of the intestine. If they belong to the small intestine, they usually more or less change their location. (For distinguishing these tumors from those of the other abdominal organs, of the peritoneum, and of the abdominal wall, see below. For inflammatory tumors of the intestine, perityphlitis, etc., see Peritoneum.)

Tumors of the rectum[1] cannot be recognized from the abdomen. Those at the point of union between the transverse and the descending colon are often recognized late because they lie concealed. They may easily be confounded with tumors of the spleen or with the kidneys.[2] In this connection we must bear in mind the phenomena of stenosis.

[1] For these, see below.

[2] See these.

For *peritoneal friction-sounds*, see Peritoneum; for *cooing sounds* that can be felt, see Auscultation of the Intestine.

Palpation of the Rectum.—The rectum must be examined with the finger if the movement of the bowels or the character of the stools indicates disease of this organ, or if disease in the neighborhood (as the wall of the true pelvis, the prostate, or the seminal vesicles in men, the uterus and its annexæ in women) is suspected. In making the examination we first obtain a view of the anus externally. The anus is to be examined for varices, changes in the mucous membrane, etc., and its neighborhood for signs of syphilis, rectal fistula, etc. Sometimes it is also necessary to obtain a thorough emptying of the bowel beforehand. The index finger is to be oiled, and introduced with the patient either lying on the side or back.[1] When the rectal sound is employed in order to reach a stenosis beyond the reach of the finger, the greatest care is necessary. It is best to employ a sound open at the end, so as to throw in some lukewarm water by means of an irrigator—a proceeding by which any obstruction to the passing of the sound may be gotten out of the way. Sometimes a large quantity of water is thus employed, as recommended by Hegar (see also the works upon surgery for the employment of the mirror in making the examination).

Distending the descending colon by inflating it with air introduced from the anus through the sound, if carefully done, is not dangerous, and is very strongly recommended for determining the location of the colon with reference to other organs, tumors,[2] the figure and condition of the colon itself. When there is a suspicion of a stomach-colon fistula, sometimes a positive diagnosis may be made by this method if it is noticed that the stomach unmistakably presses forward in connection with the colon. If this phenomenon is absent, a stomach-colon fistula cannot with certainty be excluded, because the passage of air from the colon into the stomach may be absent if the fistula be small or its orifice closes like a valve.

Percussion of the Intestine.—Generally the intestine gives a tympanitic sound; with meteorism with great tension it may become clear, non-tympanitic. Over large intestinal loops and also over the stomach (with like tension) the sound is deeper than over narrow portions; over lax portions it is deeper than over those under strong tension. But we can hardly ever determine as to the width of any portion of intestine by the resonance, chiefly because of the influence of tension, which, for a single loop of intestine, we cannot at all control. Hence we cannot with certainty determine by percussion the boundary between the colon and small intestine, a dilatation above a stenosis from another portion, or intestine from the stomach. At most, we can only determine the boundary of the descending colon by artificially inflating it.

(For determining by percussion the boundaries of the abdominal organs that do not contain air, see under the different ones.) Intestinal tumors do not always become so large as to give dulness. In percussing them we first press tolerably deeply with the finger used as a plexímeter, and if we do not find dulness, we press still deeper, in order

[1] For examining during narcosis by introducing the whole hand, see works upon Surgery.

[2] See Spleen, Kidneys.

that we may push aside any fold of intestine that may lie over the tumor ("deep percussion"—Weil).

Auscultation of the Intestine.—Grumbling sounds and splashings, which may often be heard at a distance (*Borborygmi*), and are in themselves very troublesome (especially in women who have had children), do not have any further significance. A loud cooing is not without diagnostic value if it occurs at the close of an attack of pain like strangulation. Even if we cannot see any intestinal peristalsis, we must remember the possibility of stenosis of the intestine.

Although formerly too much importance was attached to it, yet there is some diagnostic value in the cooing, which is more frequently felt than heard in the ileo-cecal region in typhoid fever (ileo-cecal gurgling).

EXAMINATION OF THE PERITONEUM.

Pathological conditions of the peritoneum are, in part, of such a character that they affect the outer layers, the coverings of the other abdominal viscera; hence possible anomalies of the peritoneum may be overlooked in the direct examination. Thus, very many diseases of other abdominal organs are combined with those of the peritoneum. This fact and the anatomical interrelations of the diaphragm and certain other organs make it very difficult to give a separate description of its physical diagnosis. In what follows we mention what may be learned in peritoneal diseases by the separate methods of examination, but we call attention to the point that the examiner ought to learn to give his attention to all the abdominal organs, by inspection, palpation, etc., at the same time.

Inspection of the Abdomen.—In diseases of the peritoneum this may reveal *distention of the abdomen*, which may be quite considerable and quite like intestinal meteorism. *Meteorismus peritonei*—that is, escape of air into the abdominal cavity from the intestine or stomach—is a very serious condition which always results in peritonitis.[1]

There is *general*, though often unequal, *distention* when there is freely-movable fluid in the peritoneal cavity—*ascites*. Such a fluid effusion collects in the most dependent part of the abdominal cavity—first in the true pelvis; then, as the amount increases, it rises higher, reaching the abdominal wall, where its level may stand at different heights. The abdominal organs that contain air float upon the top of the fluid, so far as the peritoneal fold permits. In consequence of the increased internal pressure the abdomen is broader and the lower parts contain the fluid, while the small intestine, containing air, generally lies at the upper part and is in contact with the abdominal wall. But the fluid, since it is freely movable with every change of position of the body, always occupies the most dependent part, and, if the tension of the abdominal wall is not too great, there often results an unequal distention of the abdomen which varies with the position of the body. In the dorsal position it is quite toward the sides; when lying upon the side it is over the inguinal and lumbar regions upon each side; while in the sitting posture it fills the dependent abdominal sides, the upper

[1] See below.

portions being empty; and in standing, the lower part of the abdomen projects. If there is so large an effusion as to fill the abdomen very full, there is no change in the distention, and it is also more regular, like that we have with marked meteorism. (Regarding the high position of the diaphragm when there is distention of the abdomen, see Respiratory Organs and Liver.)

If the *skin* is examined, when there is marked effusion it will not at all look as it usually does: on account of the tension it is smooth, shining, and shows, especially in the dependent parts, a peculiar bluish shimmer. When the tension is of long standing there are colorless streaks or striæ which are formed in the skin by the continuous stretching, as in the scars resulting from pregnancy, so called from their chief cause. The umbilicus may be obliterated or even project. In marked *ascites* the cutaneous veins of the abdomen are found enlarged, since as collateral veins they must take up the overflow of the intra-abdominal veins, which are compressed. Under some circumstances there may be edema of the legs from compression of the iliac veins. (Regarding the *caput medusæ* and the abdominal veins in general in cirrhosis of the liver, see under Liver.)

Ascites that moves about generally results from transudation into the abdominal cavity from stasis, being rarely, except in the beginning of a disease, dependent upon inflammatory exudations. In the former case it is either a partial indication of general dropsy and connected with edema,[1] or entirely the result of obstruction of the portal vein (cirrhosis of the liver, compression, and thrombosis of the vein). In the latter case it is a sign of peritonitis.[2]

Circumscribed distention of the abdomen where there has been little or no change in posture may be due to inflammatory fluid exudations, which are enclosed between adhesions of the intestine to itself or to the abdominal wall, or by any kind of tumor in the abdominal cavity; and also by tumors or abscess in the abdominal wall itself. Circumscribed distention, with inflammatory redness, indicates a discharge outward of an abscess, either fecal or some other collection of pus in the abdominal cavity, or of the abdominal wall.

In diseases of the peritoneum **palpation** gives very important signs:

Pain exists in all inflammatory affections. It is usually very severe in *acute peritonitis*, sometimes so great that the slightest motion, or even the lightest covering upon the abdomen, cannot be borne. This sensibility is an important indication of peritonitis, especially in distinguishing the ordinary intestinal meteorism from peritoneal meteorism, sometimes also in distinguishing inflammatory ascites from dropsical ascites. *Circumscribed pain* may indicate a circumscribed peritonitis, as it occurs more particularly over tumors, abscess of the stomach and intestine. In chronic peritonitis, especially in tuberculosis, sometimes there is entire absence of tenderness.

Now and then in chronic peritonitis there is a *general, more or less symmetrical, hardness of the abdominal wall;* that is to say, it feels as if it were thickened. This is to be distinguished from the *general increased resistance from tension* due to marked distention of the abdomen from meteorism and ascites. Thus there is a marked differ-

[1] See this.

[2] See under Palpation, Percussion.

ence between the resistance of fluid and that of meteorism in a fold of intestine. The latter has more the feeling of an air-pillow, the former is more like a material substance.

But we recognize fluid with much more certainty by the feeling of *fluctuation*, undulation. A hand is laid flat upon the surface of the abdomen, and then the abdominal wall is tapped lightly with one or two fingers, just as in direct percussion. If both hands are used, fluctuation is found in a place where there is an accumulation of fluid, and the stroke of the wave is felt with every tap of the fingers. In this way the presence of even a small amount of fluid in the abdominal cavity can be made out with great certainty. When there is great effusion under high pressure this sign may fail. On the other hand, we may be deceived in the case of persons who have a large accumulation of fat in the abdomen by the trembling of the layers of fat, and possibly also by the fat in the abdominal cavity, especially in the omentum.

Very much increase of resistance, and thus an *indistinct fluctuation*, generally occurs when the *peritoneal fluid is encysted.*

Circumscribed hard resistance, now like a round ball and again cord-like, occurs with extremely great variations in chronic peritonitis, not alone of the tubercular variety, but also in the so-called simple peritonitis from inflammatory new formations; nevertheless, the former is usually the much more frequent condition. Particularly often in this, although sometimes also in simple chronic peritonitis, we feel above the navel a dense transverse string: the omentum is shrunken and thickened by inflammatory products. Besides, there are usually, but not always, the signs of encysted, or even of free, *fluid in the peritoneal cavity.* Exactly the same phenomena are present in carcinoma and sarcoma of the peritoneum.

There occurs in an acute way *resistance in the neighborhood of the cecum* in *typhlitis* and *perityphlitis.* Here there is generally a circumscribed globular or flattened globular tumor, usually immovable, which, at first at least, is extremely tender. It indicates a fixed mass of feces in the cecum or an inflammatory deposit upon the serous side of the cecum, or both. In inflammatory cases there remains for a long time, or even permanently after recovery, a dense spot (a scar from shrunken inflammatory new formation in the peritoneum). In inflammation of the vermiform appendix we can seldom affirm that there is a tumor.

Palpation of the peritoneum through the vagina in order to discover whether there are tumors, exudations in Douglas's space and anywhere in the neighborhood of the uterus, especially the different forms of peritonitis, belongs to gynecology.

Measuring the Circumference of the Abdomen.—It is not necessary to measure the circumference of the abdomen for establishing a diagnosis, but yet it is valuable for the purpose of observing the course of an abdominal affection, and particularly for ascertaining the increase and diminution of fluid exudations. It is generally sufficient to measure the abdominal circumference across the navel and the lower lumbar vertebræ. It is better also to measure the distance between the xiphoid process and the symphysis pubis.

Percussion gives valuable information regarding the peritoneum as

to whether there is fluid effusion in the peritoneal cavity, its location and nature. By percussing with some force at what we suppose to be the boundary-line we can easily determine the boundary between the dulness of fluid and the tympanitic resonance of the intestine, but we can never distinguish it from that of those organs that do not contain air, as the liver, spleen, etc. The superior surface of a freely-movable effusion is always horizontal, and hence its upper boundary-line must correspond to a section of a horizontal plane drawn through the abdomen in whatever position the patient may assume. Whenever the patient changes his position, the effusion immediately changes its relations to the abdominal cavity.[1] Hence the result of percussion changes with the position of the body: if the patient lies upon the right side, then the portion of the abdomen which is now lowest gives a deadened sound, the upper boundary of which is horizontal; in the left half of the cavity there is tympanitic resonance; if the patient turns upon the left side, this is now dull and the right is tympanitic. This is an important sign, not only that the fluid is movable, but often that there is fluid present. Small effusions, which rarely rise only a little above the pelvis, will hence be first recognized by percussing when the patient stands upright. If there is then dulness above the symphysis pubis, it immediately disappears when the patient lies upon the back. Very large effusions may fill the abdomen so full that the intestines, on account of a short mesentery, cannot float, and hence cannot come in contact with the abdominal wall. Then the strongly-distended abdomen gives a dull sound throughout, and we sometimes notice a change of the boundary of dulness only in the position on the side, when the upper portion gives a clear sound.

When the *fluid moves about with difficulty*, slowly and incompletely changing its location with the change of position of the body, and still more if it is entirely immovable, *inflammatory exudation* with gluing or adhesion of the intestines together and to the abdominal wall is indicated. If the fluid does not move, it is said to be *encysted*. But not infrequently even inflammatory exudation, at least in the beginning of its effusion, is freely movable.

According to F. Müller, 200 c.c. of ascitic fluid can be demonstrated with certainty in children, while with 150 c.c. there is uncertainty, and 100 c.c. cannot be recognized at all. In adults only 2000 c.c. give distinct dulness, which changes with change of position, whilst with 1000 c.c. of liquid the result is doubtful.

Percussion may be an important aid in recognizing *meteorismus peritonei*, in so far that in many cases, if adhesions have not already been formed before the occurrence of perforation, it gives a perfectly uniform tympanitic or, if the tension is great, a non-tympanitic, sound over the whole abdomen, also over the region of the liver and spleen, and, besides, on account of the diaphragm being arched greatly, as far as the fifth, or even the fourth, rib. Not infrequently in this way we obtain Heubner's rod-pleximeter phenomenon [see page 117].

Subphrenic peritonitis, pyopneumothorax subphrenicus (Leyden), *subphrenic abscess*. We understand by this an ichorous-purulent, sacculated peritonitis below the diaphragm. From paralysis the dia-

[1] See above, under Inspection.

phragm is pushed very high into the thorax, causing a marked retraction or compression of the lung of that side. That half of the thorax is broadened, and by the presence of pus and gas in the cavity one is apt to mistake the condition for pyopneumothorax. Peritonitis of this character usually begins at the stomach as an ulcer, or at the intestine, especially at the vermiform appendix and cecum. In making a differential diagnosis we observe whether, in the *status præsens* or in the previous development, there were indications of disease of the lungs or, on the other hand, of the abdomen, and also whether the lung of the diseased side still performs the motions of respiration. During puncture it has frequently been found that during inspiration the pressure in a subphrenic cavity rises, while it falls, of course, in a pleural cavity. This can be recognized by the varying rapidity of discharge from the aperture made by the needle or by introducing a manometer into the cavity.

The presence of air which has escaped into the peritoneal cavity is shown in many cases by the *clear*, *metallic ringing*, *intestinal sound* in the upper part of the abdominal cavity, sometimes even a *metallic*, *transmitted breathing sound* which it yields to *auscultation*. Moreover, with the inflammatory deposits upon the reduplications of the peritoneum, especially over the liver and spleen, there occurs synchronously with breathing a peritoneal friction-sound exactly corresponding to the pleuritic friction-sound. It is very rarely produced by peristalsis over the intestines. If the friction-sound is pronounced, it can also be felt.

When it is advisable, as a therapeutic measure, to draw off fluid from the peritoneal cavity by *puncture*, it may be of diagnostic value in two ways:

1. It is then possible to examine the organs in the abdominal cavity which previously were concealed by the ascites. Not only does the fluid prevent the examination of the organs more or less completely covered by it, but the folds of the intestine floating upon it also do so, in that they crowd in between certain parts, especially the liver and spleen and the anterior abdominal wall. When the abdomen has been emptied, its wall, which before was tensely stretched, is very lax, and this renders the examination extremely easy. Hence we can now usually very easily discover the diseases which caused the effusion (*cirrhosis of the liver*, *tumors*, which press upon the portal vein, *cancer of the stomach*, *ovarian tumor*, etc.), or certain results of peritonitis (bands of scar-tissue which compress the intestine, swollen mesentery, etc.).

2. The fluid that has been drawn off can be examined. It is as important to do this as to examine pleural fluid.[1]

The ordinary hypodermatic syringe, having a thinner and larger cannula, holding 1 gram—not the larger one recommended for puncturing the pleura—is to be employed for puncturing the abdomen. The place of puncture, the syringe, and cannula are to be carefully disinfected before the operation.

In selecting a place to puncture it is necessary to be careful to avoid the stomach and intestines, particularly when they are not

[1] Which see, p. 136, *ff.*

adherent to the abdominal wall. It is true that experience teaches that even in puncturing a free coil of intestine there is scarcely any risk, but still precaution cannot do any harm. An exploratory puncture is principally required where the question concerns the distinction of solid tumors from those containing liquid or from capsulated liquid exudates, or where we wish to learn something of the nature of a fluid accumulation. In all such cases we have to do with places that are dull on percussion, therefore where, *a priori*, the danger of puncturing the intestines is not great. Nevertheless, a puncture of the intestine may easily be made, even where superficial and deep percussion has given a dull sound. Therefore, in general, it is more desirable to refrain from puncture in the abdominal than in the pleural cavity.

Frequently the principal interest one has in an exploratory puncture is that we wish to make certain about the possible presence of pus; and pus enclosed in indurations generally causes the greatest difficulty.

The most frequent case of this kind is to determine a perityphlitic abscess. Here there is usually a moderately thick induration, and to explore it is really a puncture in the dark. A puncture of the intestine in such a case will scarcely have very critical consequences; but it is of more moment to have opened into the free abdominal cavity a road for the pus through a thin spot of fibrous adhesion. Thus it has happened that the exploratory puncture has not revealed an actual abscess, or it may show pus, but not clearly indicate its relation to the appendix and cecum. For this reason some do not employ exploratory puncture here. At all events, we advise always to make it with a fine cannula only.

As for the examination of the exudate which has been withdrawn, it is in all respects the same as that of the pleural fluids.[1] Besides strepto- and staphylococci as exciters of acute peritonitis, there are also to be considered, above all, the bacterium coli commune. In chronic peritonitis it is of supreme interest to decide whether it may not be tuberculous in its nature. Microscopical examination of the exudate and of sedimented or sterile filtrated exudate has almost no value at all, while culture or vaccination also has scarcely any. Usually nothing but an exploratory laparotomy, removing a small piece of induration, and vaccinating a guinea-pig, settles the question whether there is tuberculosis or not. However, this question is often decided indirectly—*i. e.* by the presence of other tuberculous diseases, as pulmonary, pleural, glandular, or genital tuberculosis.

Chylous ascites has been observed in some cases of compression of the thoracic duct; the ascitic fluid is, to a varying extent, milk-like in appearance. It contains molecules of fat and a ferment that forms sugar.

EXAMINATION OF THE LIVER.

Anatomy.—The liver, covered by the peritoneum, lies close to the diaphragm—within its arch—and is held in place by the suspensory ligament and by the intra-abdominal pressure exerted upon its lower surface. About three-fourths of it is in the right side of the body, and

[1] Compare p. 136, *ff.*

one-fourth in the left. With reference to its superficial topography, a larger portion of it belongs to the right hypochondrium, extending into the epigastrium, with a small portion into the left hypochondrium. Usually it does not extend so far to the left as the apex of the heart.

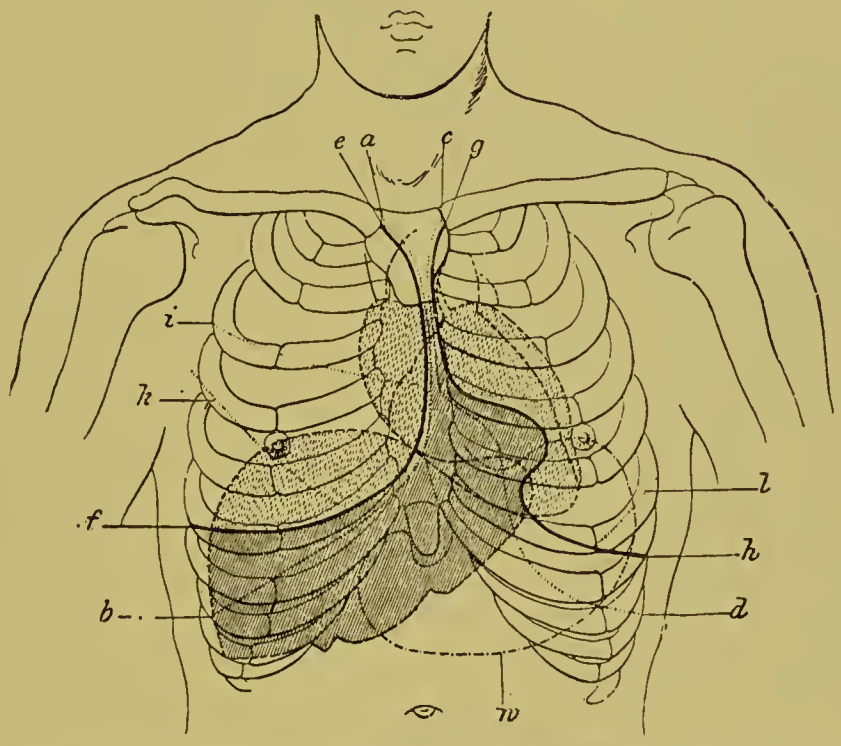


FIG. 104.—Location of the thoracic contents, of the stomach, and of the liver, from in front (Weil-Luschka).

The unbroken hatched lines represent the portions of the heart and liver that are in contact with the thoracic wall. The portions of these organs that are not in parietal contact and are covered by the lungs are represented by the light hatching: *e f* (——), border of the right lung; *g h* (——), border of the left lung; *a b*, and *c d* (. . . .), boundary of the complementary pleural sinus; *i*, boundary between the upper and middle lobes of the right lung; *k*, boundary between the middle and lower lobes; *l*, boundary between the upper and lower lobes of the left lung; *w*, stomach (greater curvature).

Above, the lungs and heart glide over it, and it glides over the stomach (see Fig. 104).

The extent to which its surface is in contact with the thoracic wall is determined by the relation of its upper surface to the diaphragm. Hence during expiration it rises in the right half of the body as high as the fourth intercostal space, and with its extreme left end to the fifth rib. The lower border, in the scapular and middle axillary line, stands about at the eleventh rib in the mammillary line, just at the border of the ribs, then proceeds obliquely upward toward the left, through the epigastrium, under the left border of the ribs, and almost to the apex of the heart. In the middle line, it stands about midway between the xiphoid process and the umbilicus. The *gall-bladder* lies just where the lower border of the liver passes under the right border of the ribs, hence close within the right mammillary line.

The *organs that border upon the liver* are the lungs, the heart, and the diaphragm above, and the right kidney, colon, and stomach below. That portion of its upper convex surface which is not covered by the lungs or heart is parietal. This parietal portion is very small behind.

As it comes forward it is much broader, and is, for the most part, covered by the chest-wall, except in the epigastrium, where it is free from its bony covering.

With children the liver is proportionately larger in all dimensions, so that its lower border is in the axillary line below the border of the ribs.

Normally, the liver, strictly speaking, only moves in connection with the diaphragm.

Inspection of the Liver.—This is made with the body in the dorsal position, moderately elevated.

In the healthy condition, in adults, absolutely nothing can be made out. The right and left hypochondriac regions are exactly alike. In small children we can sometimes notice a moderate projection of the right hypochondrium.

Projection of the right hypochondrium, or also of the epigastrium and the region below the right border of the ribs, indicates enlargement of the liver. This must be pretty well marked in order to be noticed in this way. Where the thorax is very stiff the ribs do not usually project; but when the ribs are very flexible (children, young females), where it can relatively easily take place, the projection of the abdominal wall is plainer if the abdomen is a little full and the covering thin.

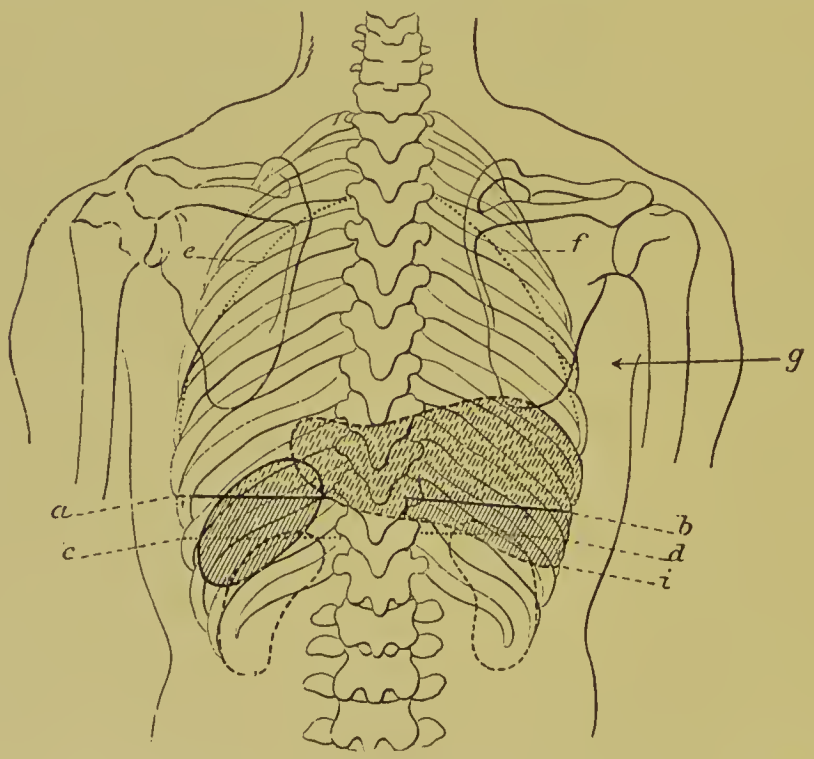


FIG. 105.—Location of the lungs, liver, spleen, and of the kidneys, from behind (Weil-Luschka).

The liver and spleen are represented by the same kind of hatching as in Fig. 87: *a b* (——), lower border of the lungs; *c d* (. . . .), complementary space; *i* (- - - -), border of the liver; *e f* (. . . .), boundary between the upper and lower lobes of the lungs; *g*, boundary between the upper and middle lobes of the right lung.

If the projection is entirely of the portion of the abdomen below the border of the ribs, it points more to a displacement of the liver down-

ward. There may be very marked distention when an enlarged liver is so displaced.

It is very rare to see or to feel *the lower border of the liver*. But it may be if, on account of enlargement or displacement, or both, it is located low down[1] and if the abdominal wall is thin. We can then also observe how the border of the liver moves downward with the motion of the diaphragm in deep inspiration. For observing this, the light must come from the head of the bed.

When the wall is very thin *tumors* on the surface of the liver in contact with the abdominal wall or on the lower surface of the border, and also a distended gall-bladder, can be seen. With deep breathing they follow the motions of the diaphragm, and they transmit the motions to tumors of the stomach or omentum, which may be adherent to them, or, like them, visible.

Finally, *arterial or venous liver-pulse* may be visible, especially the latter, which always accompanies enlargement of the liver.[2]

Enlargement of the liver may be dependent upon different diseases of this organ. In engorgement of the liver, especially in mitral defects and in emphysema, in fatty or amyloid liver, or when it is due to obstruction of the gall-bladder, and in diffuse hepatitis, in certain acute infectious diseases, the enlargement of the liver is tolerably uniform, its form being retained. It manifests itself by its lower border moving down into the abdomen, but, on the other hand, the diaphragm is pressed upward only when the liver is very greatly enlarged or when the general abdominal pressure is increased (especially in ascites). The liver is *irregularly enlarged* in carcinoma, echinococcus, generally in syphilis, and in abscess. To what extent it is noticeable depends upon the location of the swelling, whether anterior, inferior, or superior, with displacement of the diaphragm.

Downward *displacement* (*or dislocation*) *of the liver* occurs generally with depression of the diaphragm, with severe emphysema, with pleurisy or pneumothorax of the right side. Left-sided pleurisy or pneumothorax, pericarditis, though generally only to a slight degree, press the point of the left lobe of the liver downward, and thus the lower border of the liver in the epigastrium is horizontal. Moreover, under some circumstances the liver is pressed downward by subphrenic abscess,[3] which at the same time pushes up the diaphragm. Lastly, here belongs the "wandering" liver, due to relaxation of the suspensory ligament (occurring in women who have borne children). It is only in the two conditions last named that it is not in contact with the diaphragm.

It is to be observed that the lower border of the liver moves downward, not only when it is enlarged, but also when it is displaced. These two conditions will be distinguished chiefly by palpation and percussion and the consideration of the accompanying conditions of the organs in the chest and abdomen.

Displacement of the liver upward can, of course, only take place when the diaphragm is higher than normal, as in retraction of the lungs, pressure from below, inflammatory or neurotic paralysis of the diaphragm.

Palpation of the Liver.—In every relation this is the most

[1] See below.

[2] See pp. 221 and 227.

[3] See above.

important and certain method of examining this organ, and hence must be most diligently practised by the beginner. It is best to have the patient in the dorsal position and the abdominal wall as relaxed as possible. We first seize, with the warm hands, the whole abdominal sac, have the patient open the mouth and breathe quietly. Drawing up the limbs is of little aid and disturbs the examination. We very frequently make use of deep breathing, because in this way the parts hidden under the ribs move lower down, and the border or any small unevenness, etc. can be felt more distinctly as it moves against the examining fingers; and, lastly, because the liver can be distinguished from other organs (kidney, colon, omentum, often stomach, abdominal wall) by its motions during deep breathing. By *striking palpation* we understand a brusque stroke with the tips of the fingers. We employ it in meteorism and ascites in order to push aside for the moment a layer of intestine lying over the liver or fluid, and thus be able to reach the liver with the tips of the fingers.[1]

Normally, in the adult, with the ordinary thickness of abdominal wall, we can feel scarcely anything of the liver. If there is a thin, lax wall (especially in women), we not infrequently feel the edge of the liver in the mammillary line at the border of the ribs, seldom also in the epigastrium, particularly if it is pressed down in deep inspiration. In children it is often very distinct.

For example, we take a condition bordering on the normal, the so-called *constricted liver*, a disease almost without significance. It occurs in women who have laced themselves very tightly for a long time. Corresponding to the anatomical condition of the liver we can feel a tongue-like prolongation of the right lobe, which prolongation is separated from the mass of the liver by a constricting furrow close under the border of the ribs. Sometimes the constricted liver is sensitive on pressure.

In ascertaining the *pathological conditions of the liver* by palpation a series of points of view come under consideration:

1. *Tenderness.* There is no tenderness with the fatty, amyloid, cirrhotic liver, with echinococcus (if there is no formation of pus), nor engorged liver (infrequent) if it has been for a long time uniformly engorged: the syphilitic liver is usually not tender, but sometimes it is so. Generally, in the beginning of cirrhosis the liver is sensitive, also in biliary engorgement. According to the extent to which the peritoneum is involved, carcinoma of the liver may be entirely without tenderness, or it may be very sensitive; also, when engorgement of the liver has rapidly developed, it may be very tender. When an abscess of the liver is parietal, possibly involving the peritoneum, there is a circumscribed area of great tenderness; with deep-seated abscess there is no pain. Tenderness of the liver may, besides, be caused by chronic (often tubercular) peritonitis, without there being any trouble with the liver itself.

2. The *size and form.* Depression of the lower border, without change in form, indicates uniform enlargement, but possibly also displacement. Unless there is considerable enlargement it is often

[1] See, moreover, what is said on page 285 regarding palpation of the abdomen after puncture.

difficult to distinguish between these two conditions. If there is simultaneously tenderness and hardness,[1] or if there are conditions of other organs which make enlargement of the liver probable, as valvular disease of the heart with engorgement, one of the diseases causing an amyloid condition, etc., then we are very seldom wrong in the supposition that there is an enlargement. On the other hand, displacement may be made more probable, for example, by the existence of *pleuritis exudat. dextra*, etc.[2] There also may be at the same time enlargement and downward displacement. But it must be remembered that when a liver is markedly displaced downward the impression is easily made that it is also enlarged, because by traction about its transverse axis it becomes parietal to a larger extent.

When a downward-displaced liver is distinctly movable by pressure with the finger, in such a way that in the dorsal position it can be brought back to its normal position, then we have a "wandering" liver.

The *form of the liver* is recognized with varying distinctness, according to the increased extent to which it lies against the abdominal wall when it is enlarged. It has already been mentioned under what conditions the liver retains its form. Tumors of all kinds (especially carcinoma, gummata, echinococcus) and scars (syphilis) change its form. Whole portions of the parenchyma of the liver may often, not always, be marked off by the scars of syphilis if they are very deep—"lobulated liver."

3. Again, the *surface of the liver* can be judged by the portion of the upper surface or the lower border which is accessible to palpation, and we can do this best by moving the finger-tips with the abdominal wall back and forth over the liver. In individual cases it is possible to feel a portion of the lower surface. In *engorgement of the liver*, in fatty liver, in amyloid liver, in a portion of the first stage of cirrhosis, and in the so-called *hypertrophic liver*, the surface will be found to be smooth; also, in echinococcus, carcinoma, and syphilis of the liver if we palpate a portion entirely free from tumor or scars. Small inequalities, generally to a certain extent uniform over the whole palpable portions of the surface, sometimes so fine that if the abdominal wall is thick it is difficult to feel them, are the characteristic signs of ordinary cirrhosis of the liver (interstitial hepatitis, granulated liver) toward the end of the first stage and into the second. Here, for two reasons, it is usually very difficult to reach the liver with the fingers: first, because in the second stage it is smaller, and hence is to a less extent parietal; and second, because the disease is commonly associated with ascites. For this reason what has been said regarding "stroking palpation" and examination after puncture applies especially here. It is further to be remarked that the surface of the liver in chronic, and especially in *tubercular*, *peritonitis* may feel tuberculated in consequence of inflammatory growths upon the serous coat, and this without there being any cirrhosis (although not infrequently this exists at the same time). Large rough tumors, from the size of a cherry to that of an apple, often mingled with small knots, are the usual appearances with carcinoma of the liver. We can sometimes recognize upon the top of these

[1] See below.

[2] See above.

carcinomatous knots a depression, the cancer navel, but they are of neither positive nor negative diagnostic weight. More smooth, flat projections, especially if, besides, we can feel scar-like depressions, indicate the presence of syphilitic gummata. *Echinococcus* causes smooth tumors which, according to their location, are flat or elevated, or they may even stand out prominently from the surface of the liver; abscess of the liver also causes smooth prominences of different sizes and elevations.

4. The *consistence of the liver* is uniformly, and generally markedly, increased in amyloid disease, engorged liver, and in cirrhosis. Carcinoma manifests itself, as elsewhere, usually by great density. Abscess of the liver and echinococcus bladders may distinctly fluctuate; the latter often, if tightly full, feel dense as well as elastic, and we can sometimes recognize by quick, short strokes of the opposing hands a peculiar whizzing—the *hydatid thrill.*

In many cases exploratory puncture will be indicated, as, for instance, in order to recognize or exclude echinococcus or abscess. (Regarding the condition when there is echinococcus, particularly of the effects, see Tumors of the Abdomen.) Moreover, it is necessary to compare the results of palpation, in the broad sense of the word, with the accompanying appearances of other organs which belong to the individual diseases of the liver. These may have a casual relation to one another (constitutional syphilis, primary cancer of the stomach, etc.), or they may be results (ascites in cirrhosis of the liver or pressure from tumors, scars of the portal vein, rigors in abscess of the liver, etc.).

The *gall-bladder.* If this is normal, it is only in cases of extreme emaciation that it can occasionally be felt. This is much sooner possible when it is abnormally full of fluid, as in biliary engorgements, *hydrops vesicæ felleæ*, suppuration, or when it is distended with gall-stones. In biliary engorgement and catarrhal icterus it is possible to diminish the gall-bladder by carefully compressing it and expelling the contents into the ductus choledochus and the duodenum. When there are gall-stones, if the abdominal wall is thin we sometimes get the distinct impression of a sac filled with angular stones rubbing against one another. A dense, rough tumor indicates *carcinoma* of the gall-bladder.

Percussion of the Liver.—Wherever the liver is in contact with the thoracic or abdominal wall we, of course, have dullness, and this is an absolutely deadened sound where the liver receives the whole of the percussion-stroke, and the stroke is not permitted to reach to an underlying air-containing organ, as the intestine or stomach. Relative dulness, with tympanitic associated sound, occurs when a thin layer of liver lies over the stomach or intestine, as is the case in the neighborhood of the lower border of the liver. To a certain extent it depends upon the strength of the percussion-stroke whether we have a relative or an absolutely deadened sound:[1] the weaker the stroke the sooner do we have absolute dulness. The varying thickness of the covering of the liver is confusing, consisting partly of ribs and partly of abdominal wall. Still more confusing for exact examination is it that the border of the arch of the ribs, at the most important point in

[1] See p. 94.

the mammillary line, normally exactly corresponds with the lower border of the liver. The difference in sound which is caused by this change in the covering alone obscures the exact examination of the liver at this point.

The *limits of the liver*, so far as they are determined by *percussion*, are ascertained by gentle percussion at the right lower border of the lung by the transition from the clear lung-sound (or relative liver-dulness) to the absolutely deadened sound. Thus, the upper boundary

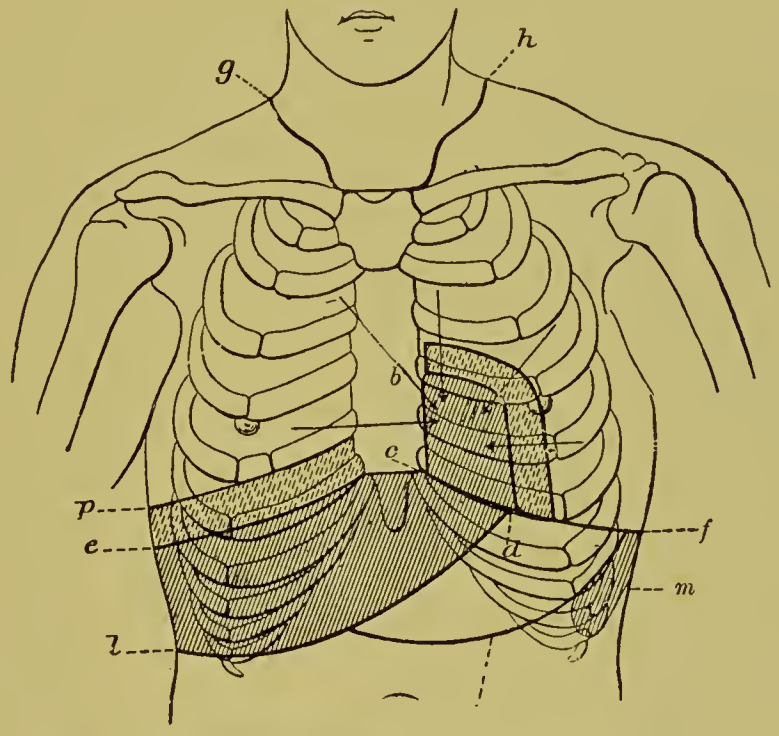


FIG. 106.—Percussion boundary of the liver in front (Weil).

g h, the upper limits of the lungs; *e f*, the lower limits of the lungs; *b d*, the boundary between the lung and heart at the incisura cardiaca. The darkly-hatched surface represents the portions of the heart and liver that are in contact with the chest-wall; the light hatching, the so-called relative heart- and liver-deadness; *m*, spleen-deadness; *n*, the average position of the lower border of the stomach.

of the parietal part of the liver is easily found, with the exception of a small portion where the liver lies against the heart (see Fig. 106). Here we cannot determine the boundary by percussion, because the heart-dulness and liver-dulness cannot be distinguished. The lower border of the liver near the spine cannot be pointed out, because it joins the kidney (see Fig. 105), but everywhere else its sound could be very easily distinguished from the tympanitic sound of the stomach and intestine if its anterior part were not too sharp; that is, if the liver were not here too thin. For this reason, even with the most gentle percussion in the epigastric region, it is usually found too high. Often no distinct liver-dulness can be perceived in any portion of the epigastrium. Moreover, we must guard against being deceived by the dulness of one of the bellies of the rectus abdominis (lax abdominal wall).

The *relative liver-dulness* lying above the absolute does not correspond to the anatomical size of the liver, which lies much farther back than this, as is shown by a comparison of the anatomical figure (see

Fig. 104) with the boundary as determined by percussion. This is because the lung becomes thinner at its lower border; moreover, it is only anteriorly and at the side that it is always distinctly present. It usually fails between the scapular line and the spine, owing to the thick wall and the diminished sharpness of the edge of the lung.

Mode of Procedure.—We percuss strongly or lightly down a known vertical line on the thorax for determining the beginning of relative liver-dulness, and thus fix the lung-liver boundary; that is, the transition from the relative to the absolute liver-deadness. Then we percuss downward, through the extent of liver-dulness, until by the gentlest percussion we get the entirely pure tympanitic sound. From this point we go again upward till we get the first indication of relative dulness. We determine the exact boundary-lines by exclusion.[1]

The *average boundary-lines of the liver*, as determined by percussion, are about as follows:

The upper, the lung-liver boundary: Middle line, the base of the ensiform cartilage; mammillary line, the sixth rib; middle axillary line, the eighth rib; scapular line, the tenth rib.

The heart-liver boundary cannot be determined by percussion, but it lies near the apex-beat.

The lower, the liver-stomach (intestine) boundary: Left of the middle line, toward the halfmoon-shaped space, ascending obliquely to about the sixth rib in the parasternal line; middle line, not lower—often higher—than midway between the xiphoid process and the umbilicus; mammillary line, at the bend of the ribs; middle axillary line, the tenth rib; scapular line, the eleventh rib.

But from these there is frequently a considerable departure, even normally. Throughout, the lower boundary has been found much higher, this being caused by a fold of intestine lying over the liver and thus diminishing the extent to which it is parietal. This is particularly the case with the ugly but not pathological form of the thorax where it is short and its lower aperture is quite wide; also in persons who have a full abdomen. In this way the liver-dulness may sometimes be entirely wanting: at the upper boundary of the halfmoon-shaped space we pass, in percussing, from lung-sound into tympanitic resonance.

Extreme elevation of the liver-dulness, although very variable within normal limits, is not at all applicable in diagnosis.

Mobility of the Boundaries of the Liver.—In deep breathing there is a more marked active displacement of the upper boundary (corresponding to the respiratory excursion of the border of the lung) than of the lower, which displacement is the expression of the movement of the dome of the diaphragm. As regards passive movements we only notice that in the left-side position both boundaries move downward —the upper distinctly so;[2] the lower, very little.

Pathological Relations.—1. The upper boundary of dulness is found higher. The cause of this can first of all be found in the pleural cavity: pleural exudation, tumors of the pleura, of the lungs, pneumonia; or in the chest-wall: tumors, peripleuritis. Then, of course, it is not possible to distinguish the dulness of what lies above the liver from that of the liver itself, since two media that on percussion give

[1] See p. 102, *f.*

[2] See Lungs.

dulness cannot be distinguished from one another. If there is exudative pleuritis upon the right side, the diaphragm is deeper and the liver moves down, causing its lower boundary of dulness to be lower, and thus in this disease there may be an extensive dulness, reaching from high in the thorax to far below the border of the ribs—dulness of the exudation plus liver-dulness.

If the conditions just named are excluded, then we may have—

(*a*) *Displacement of the liver* upward, with high position of the diaphragm. Then, at the same time, the lower border of the liver is higher, and indeed the latter is displaced upward farther than the former, because the liver as it moves upward in a sense turns on its axis; that is, the lower border turns up, so that it is to a less extent parietal—the square position of Frerichs. (For the conditions which displace the liver, see above.)

(*b*) A *tumor* of the convexity *of the liver* as a new formation, an abscess, echinococcus, when the upper boundary of dulness pursues an irregular course, according to the form of the tumor, or a subphrenic abscess. In these cases, the liver is usually displaced downward, often very markedly so; hence, the lower boundary of the liver at the same time stands deeper.

(*c*) A *general enlargement of the liver.* This causes a high position of the upper boundary only when the liver is very large. Here also the lower boundary of dulness is considerably lower. It is often very difficult to distinguish, and then only by inspection (projection) and palpation of the surface and consistence of the liver and other evidences of disease referred to under (*b*).

2. The upper boundary of dulness is found lower. This occurs—

(*a*) With a simultaneous normal position of the lower boundary in slight substantive and in vicarious *emphysema.* Although in this case the lung moves down into the complementary space, and thus covers the liver somewhat more than is normal, yet the dome of the diaphragm does not become deeper.

(*b*) With simultaneous downward displacement of the lower boundary: low position of the diaphragm with the liver; marked emphysema with low position of the diaphragm; *pneumothorax.* We can have the same percussion-results with considerable emphysema and *enlargement* of the liver. Finally, there may be low position of both boundaries resulting from the low position and enlargement of the liver. This is a frequent occurrence in severe emphysema, because of the existing engorgement of the liver.

When the liver is displaced downward it easily gives the impression of being enlarged, without such being the fact, because it is often parietal for a larger area than is normal. Also, for this reason the liver-dulness is higher than it is normally on the average; especially in pneumothorax is it often distinct.

3. The behavior of the *lower boundary* when the upper is displaced has in general been already mentioned. It remains to be noticed that, when the liver is pushed down by a thoracic affection on the right side (*pleurisy, pneumothorax*), it stands obliquely; that is, the right lobe is deeper than the left, hence the depressed lower boundary of dulness stands steeper than normal, sloping from the right toward the left. On

the other hand, when we have a pleurisy or pneumothorax upon the left side or marked *pericarditis exudativa*, since the left end of the liver (*lob. sinistra*) is then alone pressed down, the lower line of dulness is found more horizontal.

With a *normal upper border* the lower boundary stands deep and reaches farther into the halfmoon-shaped space when the liver is enlarged; on the other hand, it is higher than normal, under some circumstances even until the liver-dulness completely disappears in the following conditions: (*a*) If the liver is smaller, as in *cirrhosis*, acute *yellow atrophy*, here occurring rapidly. (*b*) As happens much more frequently than (*a*), if the liver, though perfectly sound, is less parietal than normal, or is not at all so, as in those who are on the whole well—in *meteorism*, *ascites*, entrance of air into the peritoneum. In this way even an enlarged liver may elude examination. In yet two other rare cases is the liver-dulness entirely wanting—in *situs inversus viscerum* and in cases of "wandering liver." With the latter sometimes a portion of the upper surface of the liver will be found in contact with the abdominal wall farther down.

Apparent low position of the lower border occurs when there is an airless mass below the liver, as with a full colon or a large tumor of the colon, of the omentum, or of the stomach, although these are rare.

The *form of the lower border* departs from the normal when there is unequal enlargement of the liver;[1] also sometimes in marked enlargement of the gall-bladder, seldom determined by percussion.[2]

4. Relative liver-dulness is diagnostically of little interest. It is relatively high if the diaphragm rises steeply upward and inward from the thoracic wall, and very low if the diaphragm goes off perpendicularly from the thoracic wall, as in severe *emphysema*, but especially in *pneumothorax*.

All in all, percussion of the liver, when rightly performed and correctly interpreted, is of very great value. But where palpation can be employed, as is usually the case whenever the inferior border of the liver is lower than normal, it must yield to the latter method of examination, which is more anatomical and hence more exact. If the border of the liver can be felt, then we note its course upon the body by the results of palpation and not of percussion, and proceed with the diagnosis in accordance with this position.

EXAMINATION OF THE SPLEEN.

Anatomy.—The spleen, a long, generally almost oval, organ, lies in the left hypochondrium, between the ninth and eleventh ribs, in such a way that its long diameter in the dorsal position of the body lies almost exactly behind and parallel to the tenth rib. Its posterior end lies about 2 cm. from the tenth dorsal vertebra; its anterior end, normally, scarcely reaches to a line drawn from the tip of the eleventh rib to the left sterno-clavicular articulation (*linea costo-articularis*); at any rate, does not pass beyond it. The upper (anterior-upper)[3] of the two borders of the spleen exhibits one or two notches.

[1] See above. [2] For the different kinds of enlargement, see under Palpation.

[3] In what follows I designate the two borders of the spleen as "upper" and "lower," because from the topographical standpoint that always seems to me the most natural. We

The spleen lies close to the under surface of the diaphragm, in the periphery of that portion which rises sharply upward, and toward its inner lower end it covers a small portion of the upper part of the left kidney, also the colon and stomach. Topographically, with reference to the thorax, its location is as follows: Its upper third, during moderate respiration, is covered by the lung. The lower two-thirds are in contact with the thoracic wall, but it changes its relation somewhat with the position of the body by reason of the passive mobility of the border of the lung.[1] Its upper border follows the ninth rib, forms the outer boundary of the "halfmoon-shaped space," and stands at a sharp angle with the lower border of the lung (see Fig. 107), called the spleen-lung angle, whose apex in the upright position is about at the posterior axillary line, but when in the right-side position, in consequence of the movement downward of the lower border of the lung it moves somewhat forward, even as far as the anterior axillary line. Its lower border follows the eleventh rib, and for the most part bounds the left kidney.

The spleen is in parietal contact only in its lower two-thirds, but it cannot be reached by the finger, except sometimes by turning the abdominal wall under the border of the ribs.

Inspection of the Spleen.—In the normal condition, and even when greatly enlarged, inspection of the spleen gives no result. A very considerable enlargement causes a projection of the left hypochondrium and of the abdominal region obliquely inward and downward from it. When the abdominal wall is thin the border of the enlarged organ or a circumscribed swelling on its parietal surface may be seen. Then, if the upper end of the spleen has not left its place close to the diaphragm,[2] it usually plainly descends with deep inspiration.

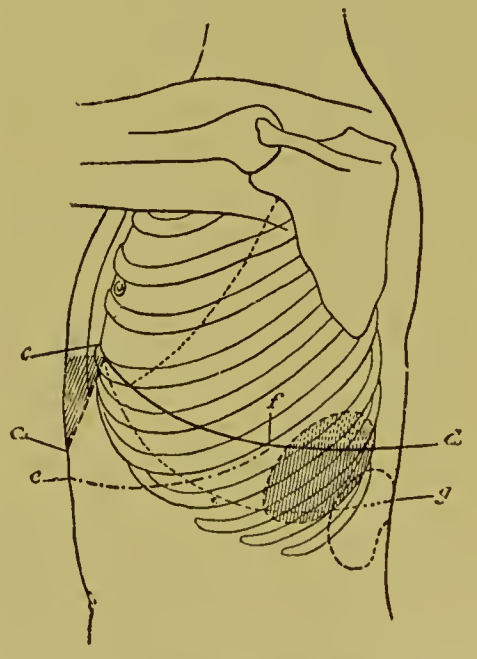


FIG. 107.—Location of the spleen (Weil-Luschka).

d, lower border of lung; *cg*, complementary space; *cf*, greater curvature. The parietal portion of the spleen, continuous hatched lines; that covered by the lung by broken hatched lines.

Palpation of the Spleen.—Palpation is very much the most important method of examination, because its results are much more reliable than is the case with percussion. Ordinarily, in order to employ palpation it is necessary for the patient to assume what is called the diagonal position on the right side—that is to say, a position midway between the dorsal and the right-side position—for the reason that percussion can be practised very much better in this position, and because the unity of the position is useful for comparing the results of the two methods of examination. When the patient is very sick it is

speak of an upper and a lower border of all the ribs, even of the lower ones, which are oblique. I cannot understand why one of the two ends of the spleen should be called the "upper" and the other the "anterior," as is done by Weil. [1] See this. [2] See below.

better to palpate in the dorsal position. When the spleen is of very considerable size this is also best (then, too, it is preferable for percussion). If it is difficult to find the spleen, then we try the right-side position, because this more fully relaxes the left side of the abdominal wall. If we have the patient take several deep inspirations, a slight swelling of the spleen can usually be made out by feeling the anterior end of the organ close to the border of the ribs, at about the tenth rib, where it comes in contact with the tip of the finger. Without further investigation we cannot refer a simple increase of resistance at the edge of the ribs to the spleen, but we must seek further to feel its borders.

The spleen can be felt—

1. In individual cases in health, when the abdominal wall is very lax; also, sometimes, in persons with deformed chest (kyphoscoliosis).

2. If it is enlarged. It may be enlarged and yet retain its form. It is uniformly enlarged in certain acute infectious diseases, as in typhoid, exanthematous, and recurrent fevers; in scarlet fever, usually in severe smallpox; in malaria, here relatively very large; in erysipelas, here often slightly enlarged; in sepsis and pyemia; sometimes in acute miliary tuberculosis; in engorgement of the spleen, especially in cirrhosis of the liver; in occlusion of the portal vein; in general venous engorgement; in amyloid disease of the spleen; in leukemia (greatest enlargement) and in splenic anemia; sometimes in infarction of the spleen (heart-disease); and also in tubercular peritonitis. We must here also mention the apparent enlargement of the spleen where there are thick peritoneal deposits (perisplenitis).

It may also be unequally enlarged by new formations, especially by carcinoma, and by echinococcus and abscess.

3. It may be felt if it is displaced, with low position of the diaphragm (rare); the "wandering" spleen.

In palpating we take notice of—

Pain.—Tenderness, probably always from the peritoneum, sometimes occurs in acute infectious diseases, in suddenly developed engorgement, in infarction of spleen, new formations, and abscesses. There may sometimes, in abscesses and infarction, be tenderness to pressure upon the ribs in the neighborhood of the spleen.

Size.—The largest tumors of the spleen, often reaching into the right side of the abdomen, occur in leukemia. On the other hand, in the acute infectious diseases we have moderate enlargement of the spleen which does not come below the border of the ribs. In other diseases the splenic tumor varies very much in size.

Pulsating splenic tumor (systolic pulsation of spleen) has been observed now and then in cases of aortic insufficiency.

Consistence.—As a rule, the consistence increases with the size, and is more dense in chronic than in acute cases. Generally, the consistence is not a guide in diagnosis.

Form; Surface.—It has already been mentioned in what diseases the spleen is uniformly, and in what unequally, enlarged. In diseases of the first group we can almost always, and in the latter sometimes, feel distinctly the notches in the upper border if the spleen projects far enough beyond the border of the ribs. In carcinoma the surface shows hard, uneven tumors; in echinococcus they are round, tense, elastic.

But in leukemia the surface is not always uniform, for it may sometimes exhibit flat elevations.

Mobility.—We have already mentioned the downward movement of the spleen with deep inspiration. I have seen cases of very great enlargement of spleen where this did not take place, because the spleen had pushed the diaphragm high up on the left side[1] and hindered its contraction.

Wandering spleen is characterized by absence of respiratory movement; but the spleen is passively movable, and sometimes even shows displacement downward with change of posture, a condition which occurs only in women. The spleen may wander astonishingly far from its place, even into the true pelvis, and it has been found in the abdominal cavity entirely free from its attachments; but usually there is only slight displacement. Tumors of this kind are recognized as wandering spleen by their form and by the notches. Often it is at the same time enlarged. A spleen displaced by the low position of the diaphragm can seldom be felt. (See further, regarding displacement, under Percussion of the Spleen.)

Relation of the colon to the spleen: Enlarged and wandering spleen lies in front of the colon. We can best prove this by inflating the colon with air[2] in connection with palpation and percussion.

Percussion of the Spleen.—*Percussion* is limited to that portion of the spleen which is not covered by the lung (Weil). It is bounded above by the lung; toward the front, superiorly, we have the upper border; inferiorly, the anterior end, and a portion some distance behind (inferior border), against the stomach and intestine: farther back, against the kidney. But this latter portion cannot be defined, there being dulness against dulness.

When we can only percuss with the patient in one position, as with very sick patients, we do so in the right diagonal posture. But if we wish to be very exact and the patient can bear it, it is best also to percuss in the upright posture. Let it be repeated that *palpation* generally, even though the physician be skilful in percussion, gives a much more certain result. But percussion must never be omitted. When the spleen is very much enlarged we may examine the patient in the dorsal position. The diagonal posture is only required to determine whether, and how much, the spleen pushes up the diaphragm.

In both the diagonal and the upright posture we begin by determining the lower border of the left lung. It is normally in the upright position: mammillary line, the sixth rib; middle axillary line, the eighth rib; scapular line, the tenth rib. In the diagonal position it varies from the seventh to the eleventh rib. From here, if we percuss in the vertical line, over the border of the lung downward, and, in the diagonal position, about in the anterior or middle axillary line, below the border of the lung, we will meet dulness instead of the tympanitic sound of the halfmoon-shaped space: *spleen-dulness.* The place at the border of the lung where the dulness is met with is the apex of the spleen-lung angle.[3] We now percuss vertically downward, through this angle beyond the deadened sound, till we come to a tympanitic (intestinal) resonance: the boundary-line is the lower border of the spleen. Then

[1] See Percussion. [2] See p. 280. [3] See Anatomy, p. 297.

we percuss from the halfmoon-shaped space and from the abdomen upon lines which cross what we suppose to be the area of spleen-dulness, and thus ascertain where the tympanitic stomach or intestinal resonance changes to dulness. This marks the line of the spleen. If we mark these points and connect them, we obtain the figure of the parietal portion of the spleen, which we can complete by determining the lower

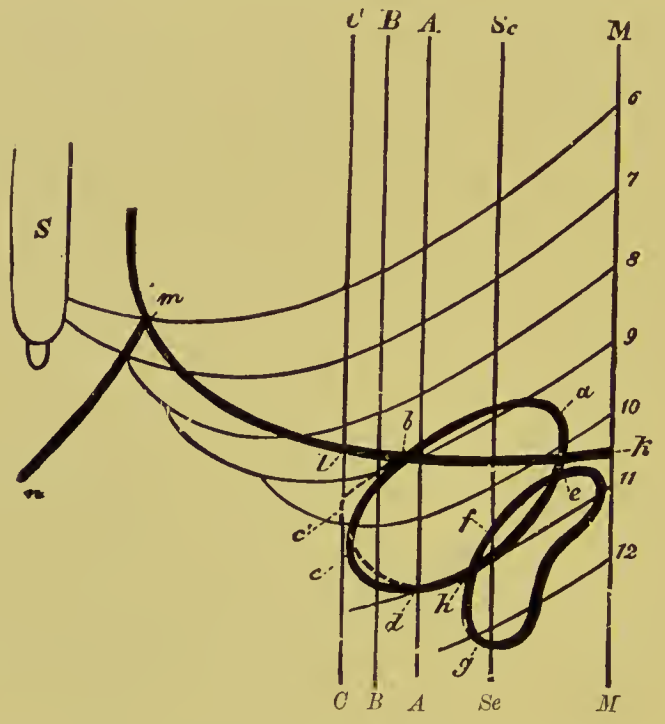


FIG. 108.—Position of the spleen (Weil).

M, the middle line of the back; *A*, *B*, *C*, the axillary lines; *Sc*, the scapular lines; *abcd*, spleen; *abcd*, unusual rhomboidal form of the spleen; *efg*, outer boundary of the kidney; *lbc*, the spleen-lung, and *dhg*, the spleen-kidney angle; *nm*, the lower border of the liver.

border of the spleen in the posterior axillary line or in a vertical line between this and the scapular line.

In the upright position the conditions are altered in such a way that the border of the lung on the left side, and with it the lung-spleen boundary, is somewhat higher,[1] and hence we find the apex of the lung-spleen angle in the middle or posterior axillary line.

As has already been said, *the size of the spleen-dulness*, with careful percussion and under favorable conditions,[2] corresponds to the parietal part of the spleen. From this we must estimate the size of the spleen. In measuring it we have only two points of departure: the height of the spleen-dulness in the vertical line passing through the apex of the spleen-lung angle, and the relation of the anterior end of the spleen to the *linea costo-articularis*. The average in health has been found to be (Weil)—

In the diagonal posture the height of the spleen is 5.5 to 7 cm., the anterior end at most reaching to the *linea costo-articularis*.

In the upright position the height is 4.5 to 6 cm., the anterior end under some circumstances passing a little beyond the *linea costo-artic-*

[1] See above.

[2] See below.

ularis, the spleen-lung angle more pointed; that is, the spleen is a little more horizontal.

We are interested in the mobility of the spleen-dulness in deep inspiration only so far as it affects the boundary between the spleen and lung.[1]

Weil has sufficiently explained why we must forego the determination of the portion of the spleen which is covered by the lung. I agree entirely with him, and refer to his work upon *Topographical Percussion.*

In the first place, we percuss tolerably strongly. If in that way we obtain no result, we then percuss very lightly. With strong percussion we very rarely have resonance over the spleen; also, with moderately strong, only rarely absolute deadness. Also, by gentle percussion, we must often be satisfied with a relative dulness associated with tympanitic accompaniment.

Departures from what has been called *the "average" in health:* [*a*] Very often in health the dulness of the spleen as regards its size or intensity is only suggested, so to speak: in such cases it is covered by intestine, or the spleen is thin and the intestines near it are distended by gas.

[*b*] The area of dulness of a normal spleen seems larger, and occasionally exactly corresponds also in form with a uniformly enlarged organ. This occurs when the stomach is overloaded with food, when there are fecal masses in the neighboring colon, when there is corpulence (the greater omentum loaded with fat), but also sometimes without these conditions being present. We must guard against deception as respects the stomach and intestine by repeated examinations, especially with abstinence from food and after free purgation. When there is obesity we ought not, on the whole, to draw any conclusion from a large area of spleen-dulness.

But, at any rate, we must never by a single examination diagnosticate a spleen-tumor from percussion alone.

Pathological Relations.—As mentioned above, *diminution of spleen-dulness* is often met with in health. In sickness it occurs from overlapping of the spleen from above by the lung: this happens with emphysema of the lung, when the lung spreads into the complementary space; sinking down of the lower border of the spleen and its anterior end is evidence of displacement downward by flattening of the diaphragm, but in emphysema this cannot be proved. There is always diminution of spleen-dulness (even to complete disappearance) when it is displaced upward, as in shrinking after pleurisy, contraction of the lung, high position of the diaphragm. Here, generally, there is no spleen-dulness at all, on account of the intestine lying over it.

Enlargement of Spleen-dulness.—If we make out such a condition, we ought to call to mind the sources of error mentioned above. We should never make the diagnosis of enlarged spleen from a single percussion without the support afforded by palpation. We must notice whether the enlarged dulness shows the relations of the figure of the spleen; if it does, then it is quite probable that the spleen is

[1] See what has been said regarding active mobility of the border of the lung.

enlarged; likewise if the examination in the diagonal and the standing position shows a similar result, with change of dulness that distinctly corresponds with the changed position of the border of the lung and the spleen.

Enlargement of the spleen is to be assumed when the vertical measurement of dulness is as much as 9 cm. or more; also if the area of dulness extends considerably beyond the *linea costo-articularis;* and, lastly, if the dulness is very decided, that is, with moderately strong percussion absolute deadness. When there is considerable enlargement of the spleen, the area of dulness upward is larger, and hence the diaphragm, and with it the border of the lung, moves higher in the chest. Moreover, in every upward enlargement of the spleen-dulness it is to be remembered that it may be merely apparent, being caused by pleuritic exudation, infiltration of the lungs, or pleural tumor.

When there is a decided enlargement of the spleen, it considerably diminishes the halfmoon-shaped space. If there is, simultaneously, tumor of spleen and liver, the space may be entirely deadened.

Auscultation of the Spleen.—In rare cases auscultation enables us to recognize peritoneal friction-sounds should there be inflammatory deposits upon the serous coat of the spleen and the parietal portion of the peritoneum opposite it, if the diaphragm is not paralyzed by the peritonitis, or the spleen has not become adherent. Peritoneal friction-sound over the spleen (and over the liver) seems to me to have greater weight as evidence that the first of the two last-named conditions is wanting than as the sign of peritonitis, for the latter usually appears to be plainer from other symptoms. It may easily happen that we find it difficult to distinguish whether we really have peritoneal rather than pleuritic friction-sound. Auscultating with the stethoscope enables us to localize the sound more exactly. We must also take into consideration the whole picture of the disease.

EXAMINATION OF THE PANCREAS, OMENTUM, RETROPERITONEAL GLANDS.

The **pancreas** is accessible for examination, and even to palpation, if it is the seat of new formation, as of carcinoma, especially of the *caput pancreatis*, and hence is larger and harder than normal: we have a roundish tumor in the right epigastrium which does not move during respiration, about midway between the point of the xiphoid cartilage and the umbilicus, hence directly under the border of the liver; or a somewhat longer tumor across the epigastrium. Unless there are characteristic associated symptoms (compression of the ductus choledochus and pancreaticus, biliary engorgement, and change in the character of the stools—diabetes mellitus), the diagnosis of tumor of the pancreas can scarcely be made from such a tumor, which may also belong to the omentum, but especially to the retroperitoneal glands.

The **omentum** also is only perceptible when it is thickened by inflammation or by new formations or by both. It frequently shrinks up to a transverse band, crossing close above the umbilicus if it is the seat of tuberculosis, or also in "simple" chronic peritonitis. As regards its mobility in respiration, such an omentum may behave differently, de-

pendent upon its being adherent to the abdominal wall or attached to the stomach. Carcinomatous knots in the omentum are best to be distinguished from similar deposits in the anterior wall of the stomach by examining the latter both when empty and when full or inflated. Sometimes it is very difficult to distinguish them from carcinoma of the liver, especially if the omentum, from adhesion with the liver, moves with each respiration. Echinococcus of the omentum is quite rare.

Enlargement of the **retroperitoneal glands** generally occurs in secondary carcinoma as firm, immovable bunches which are located in the cavity of the abdomen, about on the level with the umbilicus, but sometimes they reach even deeper. They may compress the side of the inferior vena cava or the iliac vein. These glandular tumors may easily be confounded with aneurysm of the aorta, especially if they are round tumors and propagate pulsations, and they also may even communicate a humming murmur of stenosis from the aorta.

We must again call attention to the importance of always emptying the intestines and bladder, and artificially inflating the stomach and intestines, in all cases of this character where the diagnosis is difficult.

This is not the place to explain the differential diagnosis of a large number of other affections of the abdomen, especially tumors of the uterus, ovaries; also pregnancy. (We refer for these to works upon Gynecology and Obstetrics.)

EXAMINATION OF THE CONTENTS OF THE STOMACH.

In general we may in two ways obtain the contents of the stomach for examination: when the patient vomits, or when, by emptying the stomach by means of an esophageal catheter, we remove a portion of its contents. The catheter may be introduced for therapeutic purposes or only for the purposes of diagnosis.

The latter way of obtaining some of the contents of the stomach, it is readily seen, is the more exact for making a diagnosis, because we regulate the time for doing it by the object we have in view. First, with reference to the most important problem in the diagnosis of the contents of the stomach—namely, the examination of the stomach-digestion and the secretion of gastric juice—it is only necessary to empty the stomach to obtain the object required. At the same time, it is to be remembered that in many cases the examination of vomited matters has its particular value, and that occasionally—for instance, in cases of poisoning—the fluids employed in rinsing out the stomach are to be examined if such rinsing out is therapeutically necessary.

However, very frequently it is possible to combine the therapeutic with the diagnostic emptying of the stomach, as is shown by a comparison of therapeutic indications with those of the diagnosis. We cannot here enter more fully into this subject.

Artificial emptying of the stomach, or removal of some of its contents for the purposes of diagnosis, is, as has been said, the only method which enables us to form a reliable opinion regarding the gastric secretion and the process of digestion, for the reason just given, that such an opinion can usually only be formed when the contents of the

stomach have been obtained in a perfectly unmixed state and at a definite time after partaking of a meal. Vomiting can make the artificial emptying of the stomach unnecessary only when it occurs at exactly the time desired, and when the material vomited does not contain bile and not too much mucus.[1]

Induction of emesis is contraindicated when there is a tendency to hemorrhage, and in poisoning where we have reason to think the poisons, as acids and alkalies, have caused erosion of the esophagus or stomach. Sounds, even soft ones, are to be employed with the greatest caution, and only when the stomach is unquestionably full, if there has ever been any hemorrhage of the stomach, and also when there is any suspicion of an ulcer of the stomach or of a markedly ulcerating carcinoma. It has already been mentioned[2] that a suspicion of aneurysm of the aorta forbids any introduction of the stomach-tube.

The examination of the contents of the stomach has a manifold diagnostic value. By its aid we are able to diagnose a number of anatomical diseases of the stomach before the respective conditions make any other objective symptoms. There are other diseases of the stomach which otherwise never furnish any distinct symptoms at all: they can only be recognized by an examination of the process of digestion. On the other hand, certain nervous dyspepsias are distinguished by a normal digestion which is in contrast with the severe subjective complaints. But even where a disease of the stomach—cancer of the pylorus, for instance—has been diagnosed with certainty by an external examination of the abdomen, a clear insight into the disturbances of the functions of the stomach which are caused by the anatomical lesion can only be obtained by a study of its contents. And in all these cases the examination furnishes valuable indications for treatment.

EXAMINATION OF THE PROCESS OF DIGESTION.

Preliminary Remarks upon Stomach-digestion and its Disturbances.—I. Physiologically, the stomach fulfils a threefold task—

(*a*) It initiates the alterations of the starches and albuminous portions of the food into absorbable substances, and in a lesser degree also completes them.

(*b*) It protects its contents for hours from fermentation and putrefaction during their stay within it.

(*c*) It discharges its contents within a certain time, partly (but to a comparatively very small extent) by absorption, principally in the following way: first, the fluid, and later the solid, elements of its contents pass in separate portions through the pylorus into the duodenum.

After a meal containing albumin and starches has been taken (we disregard fats, because they are not acted upon by the stomach), first there is a transformation of the starches, partly amylolytic, partly diastatic, produced by the fermentative action of the ptyalin of the saliva. Starch is converted into maltose or dextrose and achroo- and erythro-dextrin. Any cane-sugar that has been taken is inverted into

[1] See below.

[2] See p. 265.

dextrose. These processes go on rather rapidly, but are, however, usually interrupted before completion by the commencing hydrochloric-acid acidification of the stomach-contents. Simultaneously, various micro-organisms—schizomycetes, which enter from the mouth and are always present in the stomach—produce a partial lactic-acid fermentation of the sugars present there, which are capable of fermentation: hence lactic acid is formed.

This *amylolytic period* of digestion lasts a short time, varying in length according to the size of the meal; on the average, it lasts three-quarters of an hour. As a matter of course, it is entirely absent if only meat is eaten; then also there is no lactic acid.

Immediately after food is taken the mucous membrane of the stomach begins to secrete muriatic acid and pepsin or propepsin,[1] and the stomach-juice mixes with the alkaline chyme. But at first the muriatic acid is in combination, but after a period of time of variable length, on the average one-half to three-quarters of an hour, we have free muriatic acid. The amylolytic period is brought to a close, because the free acid destroys the ptyalin of the saliva. The lactic-acid fermentation too is soon afterward suppressed—*i. e.* as soon as the free hydrochloric acid of the gastric juice amounts to about 0.1 per cent. (to 0.07, according to some; to 0.12 to 0.16, according to others), and now there begins the swelling and defibrination of the meat by the hydrochloric acid, and the hydrolytic splitting up of the albuminous substances by the enzym of the gastric juice, the pepsin. At the same time the bacteria are destroyed or made ineffective.

We must now keep clearly in mind that the gastric juice secreted by the mucous membrane of the stomach—that is to say, by the glands of the fundus, which is strongly acid and contains hydrochloric acid and pepsin—acts on those parts of the gastric contents which are in contact with the walls of the stomach; moreover, that the hydrochloric acid, diffusing into the parts of the gastric contents which lie farther away from the walls of the stomach, enters into combination with these. Therefore, we must assume that there is a peptonizing and disinfecting action of the hydrochloric acid on the masses which are carried along the walls of the stomach by its peristaltic movements, before the entire contents of the stomach (or a sample taken from it) contains any free acid.

Therefore, the stomach-contents do not contain any free acid in the first stage of the secretion of the acid gastric juice, because the secreted hydrochloric acid is then in combination. In the first instance it is a fixed combination, in the ordinary sense, up to the neutralization of the existing alkali. This is followed by a looser combination with albumoses and peptones in the formation of substances which may be called acid albumins. Farther, there is a loose combination with leucin, tyrosin, and also with salts, particularly phosphates. Combinations of hydrochloric acid have an acid reaction, but pepsin has no digestive power if in contact with them. Only when the loose combination of hydrochloric acid with those substances is completed, and, besides, the stomach has gotten rid of a considerable part of the acid-dissolved albuminous substances, does free hydrochloric acid appear, and from

[1] See below.

that time on it increases to a maximum, which in health is about 2 per cent.

As by far the greatest part of the loosely combined hydrochloric acid is combined with albuminous substances, the time of appearance of free hydrochloric acid in the healthy depends, in a high degree, on the quantity of albuminous food taken. After a meal poor in albumin free hydrochloric acid is found in the second hour of gastric digestion; after a meal very rich in meat—after five-tenths of a kilogram of meat, for instance—it is found only in about the fourth hour (Moritz).

The loosely combined hydrochloric acid, which does not any more come into consideration for the peptonization of albuminous substances, is called "masked hydrochloric acid," because while it reddens litmus-paper it does not give any certain reactions of color essentially belonging to hydrochloric acid. We may approximately say that the quantity of this loosely combined acid plus the free hydrochloric acid corresponds to about the quantity of acid which has been secreted in the course of digestion.

About synchronous with the secretion of hydrochloric acid the rennet ferment (*milk-curdling ferment*) is secreted by the gastric mucosa, by which ferment the lactalbumin—*i. e.* the albuminous substance which exists in human milk and in that of mammals—is changed into its albuminate, casein, and thus made accessible to further hydrolytic decomposition. The secretion of the milk-curdling ferment seems to go on parallel with that of hydrochloric acid and pepsin in such an equable manner that in practice we need not give any special consideration to these bodies.

Pepsin and also rennet-ferment are not secreted as such by the mucous membrane of the stomach, but are formed by their zymogens [ζύμη, ferment], propepsin and rennet-zymogen. Both, under the influence of the muriatic acid, become transformed into pepsin and rennet-ferment. The lactic acid, although in very much larger quantity, has this effect upon the zymogens also.

In this second period, the hydrochloric-acid period of digestion, the important antiseptic reaction of the hydrochloric acid of the gastric juice upon the chyme also comes into consideration. Under its influence by far the greater part of the micro-organisms which have been swallowed with the food and drink are destroyed, or made ineffective for some time. This is especially true of all the different kinds of bacilli which cause lactic-acid fermentation, and the schizomycetes, which produce alkaline fermentation or putrefaction, and finally the bacillus of butyric-acid fermentation, which exists in some foods (different kinds of cabbage, peas, cheese, etc.) and several kinds of yeast. All these fungi are found in the stomach of the healthy. They become excessive in amount if the secretion of hydrochloric acid is deficient. Of the fungi which abnormally get into the stomach, the pathogenic ones are of particular importance. Of these we know that the bacillus of cholera and of typhoid fever are made ineffective or destroyed by a gastric juice whose acidity is due to hydrochloric acid, which perhaps does not happen not only when free acid can be found in the stomach, but even before that time, so long as the acid is yet in combination, because the acid which appears continually anew on the surface of the mucosa,

the nascent acid, can react on continually changing parts of the gastric contents even before it has become combined.

During the time when the food is in the stomach the reaction of the ferments upon it is very essentially enhanced by energetic peristaltic movements, which, commencing about half an hour after a meal, break up the food and mix it with the ferments, or else bring all parts of the chyme, each for a brief time, in contact with the stomach-walls and the comparatively concentrated solutions of the secretions which pour out of the walls.

The other, just as important, side of the motory performance of the stomach is that soon after taking food it commences to absorb the liquids and the more solid contents (Moritz), in that the former are transported to the portio pylorica, and from this are squirted rather energetically into the duodenum by rhythmical opening and closing of the pylorus. The remaining gastric contents are continually concentrated by the sorting of the solid and fluid contents; they are, however, also continually diluted anew by the secretion of water from the mucosa of the stomach (v. Mering). The stomach therefore provides for a continual dissolution of its solid contents by secreting water into the chyme, transporting it to the portio pylorica, and ejecting it. Finally, it ejects also the insoluble, solid residue of the meal through the pylorus.

Absorption.—Water in only small quantities is absorbed by the stomach: the freer it is from dissolved constituents the more completely is it transported, portion by portion, into the duodenum. On the contrary, according to von Mering's investigations, the walls of the stomach absorb the following: alcohol in considerable quantities; the different kinds of sugar in moderate quantities, and that better in alcoholic than in watery solutions; dextrin and peptone in small quantities; and carbonic acid is abundantly absorbed. In general the absorption is greater the more concentrated the solution; however, the more the stomach-walls absorb the more they secrete water into the stomach.

A few minutes after the introduction of water into the stomach of a dog its evacuation into the duodenum in single portions begins. Similarly, according to Leo, the milk in the stomach of a nursling is passed on.

Pepsin has no antifermentative effect, but acetic-acid fermentation is suppressed by traces of free hydrochloric acid—lactic-acid fermentation by 0.07 per cent. of free hydrochloric acid. Hydrochloric acid in combination with peptone does not prevent fermentation, as it has only a weak digestive power.[1] But it has been demonstrated in regard to the stomach of nurslings that fermentation is prevented even if no free hydrochloric acid can be found (Leo). This may be perfectly understood also as regards the processes in adults if there is kept clearly in mind the mechanical conditions which have been mentioned several times—the continual bringing to the stomach-wall of successive parts of the chyme, where they are mixed with comparatively concentrated portions of nascent hydrochloric acid. Only by gradually penetrating into the whole of the chyme does the hydrochloric acid likewise gradually become combined. But in the mean time it has already exer-

[1] See above.

cised its antiseptic and peptic power on those particles of food which are in contact with the stomach-wall.

Until now, however, there has been unsatisfactorily appreciated the greatly significant circumstance that the different kinds of food, according to their physical and chemical properties, exercise a very different degree of stimulation of the secretion of hydrochloric acid and the motor activity of the stomach, both of the healthy and of the diseased organ. This fact forms one of the principal reasons why we cannot make our examination after every kind of meal if we wish to gain the most perfect possible insight into the work of the stomach. It is necessary rather to introduce into the stomach a mixture of food the quality and quantity of whose constituents are exactly determined, thus to procure in this respect at least equal conditions for successive experiments. A meal given for the purpose of making an experiment in digestion is called a "test-meal."

II. About six hours after a mixed meal of moderate quantity, much sooner after a smaller one, the stomach has become entirely empty or at most contains only small particles of food. In the interval until the next meal, in the great majority of healthy persons, it appears that the stomach contains a very scant amount of clear fluid, with a neutral reaction, but no muriatic acid or pepsin.

The stomach-digestion of nurslings has as yet been very little studied. According to Leo, the fasting stomach of a nursling almost always contains free muriatic acid, while during digestion free muriatic acid cannot at all, or only after an hour, be demonstrated; this is not because there is none secreted, but because it is neutralized by the milk. Leo always found rennet-ferment, excepting in one case where there was rennet-zymogen. After half an hour the greater portion of the milk has passed into the intestine, and in one or at most three hours the stomach is empty. Leo also thinks that the peptonizing of the milk in the stomach is a subordinate process. He regards the stomach as really a milk-reservoir, and as offering a barrier to pathogenic micro-organisms.

III. The chief points in regard to the effect of pathological disturbances of the gastric secretion, of the motions of the stomach upon digestion, and the sterilization of the food and its further transportation into the intestine are as follows:

Diminished secretion of hydrochloric acid and pepsin, which always goes parallel with it (*hypacidity*, *subacidity*), interferes with the swelling of the meat and the peptonization of the albumin, and also lowers the antifermentative action of the stomach upon its contents. The alteration of the starches might be normal, but, at any rate, it is almost always disturbed, because, in consequence of the scarcity of hydrochloric acid, lactic acid is increased, and ultimately also butyric-acid fermentation, which quickly acidify the contents of the stomach even in a higher degree than takes place in normal digestion by free hydrochloric acid. If lactic acid be very abundant, it may even peptonize the albuminous substances if pepsin, of which, as is well known, only a very small quantity is needed, is not too much diminished. The examination of the gastric contents at the height of digestion shows diminished free acid—*deficiency of hydrochloric acid.*

If at the height of digestion free hydrochloric acid be entirely absent, we call the condition *anacidity*. The condition is one of deficiency of hydrochloric acid, but not necessarily of complete absence of its secretion. In acidity the disturbances above mentioned are present in a higher degree.

Considerable fermentation, or even *putrefaction*, develops in these cases, however, only when the chemical disturbance is combined with considerable lowering of the *motor activity* of the stomach, which certainly is very frequently the case. Only when the gastric contents for some reason remain abnormally long in the stomach is there time for fermentation to take place. However, *stagnation* of the contents of the stomach alone, without diminution of the secretion of hydrochloric acid, may have this effect, because it may come to the point that the hydrochloric acid, in spite of its abundant secretion, is not sufficient to acidify the great quantities of material stagnant in the stomach. There is then *hypacidity*, and, following this, abnormal fermentations, notwithstanding abundance of hydrochloric acid. It is often difficult to make a correct diagnosis in these cases.

Increase of hydrochloric acid, *hyperacidity*, *superacidity*, causes a too early appearance of free hydrochloric acid, and hence shortens the amylolytic period of digestion; and thus there are often, though not constantly, found during the whole period in which the food remains in the stomach physically unaltered portions of amylaceous food or larger quantities of unsplit starch—*i. e.* of starch which becomes blue when mixed with iodin. If the free hydrochloric acid exceeds 0.2 per cent. (and not infrequently it is as high as 0.4 per cent., sometimes even higher), the albuminous foods are peptonized as soon as, or sooner than, normal and more completely; that is, the period of digestion is frequently shortened. The gastric contents are often strikingly thin and clear, perhaps in consequence of increased secretion of water from the walls of the stomach; the meat is more or less changed; the hydrocarbons are sometimes scarcely changed; organic acids are absent or sparingly present.

Some conditions of hydrochloric-acid hyperacidity, with accelerated evacuation of the contents of the stomach into the intestines, change after a time, by a motor paralysis of the stomach, into a condition of stagnation of food, followed by catarrh of the mucosa with hypacidity; then the whole process is reversed.

Diminution of hydrochloric acid generally seems to go parallel with a diminution of pepsin and milk-curdling ferment. On the other hand, this parallelism is generally not present when there is hyperacidity; with increase of hydrochloric acid there even appears to be an absence or deficiency of pepsin. Some cases, where the peptonization of the albuminous substances is diminished notwithstanding the increased free hydrochloric acid, can only be explained by these facts.

It has been said above that the disturbances of the secretion of gastric juice are not seldom combined with *diminution of the motor activity of the stomach*; then, without exception, the food remains in the stomach. Both the chemical and motor anomalies are sometimes the consequences of the same cause, as, for instance, gastric catarrh; or the weakness is the direct consequence of abnormal chemical processes,

which is probably sometimes the case in hyper- as well as hypacidity. Frequently in such case, too, the food is kept back by *spasm of the pylorus*. Where there is only hypacidity with abnormal fermentation and stagnation, the latter furthers the abnormal processes of decomposition in a very disagreeable way. The more severe of these conditions are those where the stomach does not become empty from evening till morning—where, therefore, an introduction of the sound into the fasting stomach still brings forth food-material.

But the most important alterations are generally seen when for some reason or other there is *primary interference with the transit of the food*, as is the case in chronic stenosis of the pylorus from carcinoma and cicatrices. At first the stenosis of the pylorus for a time is compensated by hypertrophy of the muscular coat of the stomach, exactly as stenosis of the aorta is compensated by hypertrophy of the left ventricle. But by and by the compensation no longer keeps pace with the progressive narrowing of the pyloric orifice, and stagnation of the stomach-contents takes place: gastric digestion is retarded; there is *ectatic dilatation of the stomach;* it is no longer empty in the morning after fasting. Furthermore, in spite of abundant, and sometimes probably even increased, secretion of gastric juice, the hydrochloric acid is used up by a loose combination with the albuminates, which remain in great quantities; the free hydrochloric acid decreases and may gradually entirely fail: there is a deficiency of hydrochloric acid, as in hypacidity, or it may be called *relative hypacidity*. Lactic- and butyric-acid fermentation takes place, the more because the remaining food has time for these processes; or even alkaline fermentation or putrefaction develops. This abnormal decomposition of gastric contents injures the mucous membrane, already weakened by stretching of the walls of the stomach; indeed, it results in absolute decrease of secretion of the gastric juice.

The stomach is never emptied, although from time to time it gets rid of a part of its contents by vomiting. Besides slightly altered meat it contains unchanged hydrocarbons, diminished (or no) free hydrochloric acid; there is diminished secretion of (*i. e.* free, plus loosely combined) hydrochloric acid; the stomach contains much lactic, butyric, acetic acids, alcohol, gases (CO_2, hydrocarbonates, H_2S, etc.), schizomycetes, yeast, generally also sarcina.

In carcinomatous stenosis of the pylorus the secretion of acid gastric juice decreases particularly early, probably in consequence of derangement of metabolism by the carcinomatous condition; hence we very early and almost uniformly miss the free hydrochloric acid, and there is considerable deficiency of hydrochloric acid, even if that which is loosely combined be included.

Gases in the stomach-contents, however, need not have their origin in fermentation alone: an abnormal amount of air may be swallowed ("Luftschlucken"). Stomach-gases formed by fermentation are mostly either carbonic acid or hydrocarbonates. In the former case, if conducted through lime-water, they make it turbid; in the latter they are inflammable. When the gas is due to fermentation of the gastric contents, they show fine foam-bubbles, while from swallowed air this is not the case.[1]

[1] Compare p. 314.

From the statements now made there result the following leading points of view for the investigation of the function of the stomach:

Far in the foreground of diagnostic interest stands the question: How long does the food remain in the stomach? An answer to this is, in the first place, obtained by introducing a stomach-sound in the early morning before breakfast. Normally it should contain only a few fine uncharacteristic flocks and a non-acid, possibly also no mucous, material. If distinct coarser remnants of food are found, the motor-work of the stomach is considerably at fault, and the extent of this defect is shown by their age and the degree of their abnormal decomposition. If only the normal contents mentioned above are found, there still may be a certain retardation in the evacuation of the stomach, as the examination takes place from ten to twelve hours after the preceding meal. According to the indications of other phenomena, it will be necessary to make a still more exact investigation by the aid of a *test-meal.*

Sometimes the fasting stomach in the morning contains hydrochloric-acid gastric juice without remains of food—*hypersecretion, continuous secretion.*

If thicker quantities of mucus are present, it indicates *catarrh of the stomach.*

The examination of the contents of the stomach in the morning has the advantage that with it may be combined a therapeutic washing out.

A definite opinion regarding the length of time food remains in the stomach and the chemistry of stomach-digestion, particularly regarding the secretion of gastric juice, can only be obtained by administering a test-meal with the stomach entirely empty, and then determining the time when it has completely disposed of the food, as well as its chemical nature at the height of the gastric digestion (that is, when the hydrochloric acid attains its maximum percentage or must have attained it).

From what has been said above, it will be seen that it is desirable that the test-meal should contain a minimum of fat or no fat at all, since fat is not changed in the stomach. It is further to be remembered that albuminous substances, by forming a loose combination with hydrochloric acid, cover this acid even though it may be abundantly secreted. The secretion of hydrochloric acid is of the first importance, and, in order to form an opinion about it, it is necessary to omit albuminous material as much as possible from the test-meal, or to make use each time of exactly the same amount of this material, so as to be able from one experiment to another to make allowance for exactly equal amounts of masked, loosely combined acid.

Ewald's test-meal avoids the difficulty just mentioned in that it contains almost no albuminous material at all. It consists of two cups of tea without milk or sugar, and thirty to seventy grams of dry wheat bread, which must be thoroughly masticated. It also has the advantage that the experiment is completed in a comparatively short time. The maximum of hydrochloric acid is attained normally one hour after the meal is taken, and after two hours and a half the stomach is empty. However, this meal has the disadvantage that upon some stomachs it

exerts very little stimulation, particularly on their secretion of gastric juice, for we must assume that it is albuminous substances which chiefly stimulate its secretion. On the other hand, it imposes a comparatively slight task upon a stomach whose motive force is insufficient; and accordingly the results may be comparatively too favorable as regards the motor activity of the stomach, for we must know how the organ disposes of amylaceous as well as of albuminous substances in order to understand how it performs its function.

For this reason we have lately preferred *the test-meal of meat and starches.* Of these several have been suggested. That of Leube consists of a plate of water soup, a moderate amount of beefsteak, and a little white bread. Kussmaul's test-meal, mentioned several times by Fleiner in his writings, seems to us to be more practical still: 250 gm. of strained barley soup, 200 gm. of beefsteak, 200 gm. of mashed potatoes. Here the amount of albumin is somewhat regulated by the weight, and a similar arrangement of the conditions of the experiment seems to me to be indispensable.

Normally, Leube's test-meal has left the stomach after seven hours at the latest, often after five or six hours. The maximum of hydrochloric acid is reached about one to two hours before the time of complete evacuation of the stomach.

After Kussmaul-Fleiner's test-meal the stomach is usually empty somewhat earlier, after five to six hours, and the maximum of hydrochloric acid occurs three to three and a half hours from the time of taking the meal.

Evacuation or washing out the fasting stomach in the morning and the introduction of a test-meal one or more successive days, with attendant examination of the duration of digestion and examination of the gastric contents at the height of digestion, are almost always sufficient, combined with the other symptoms, to form a correct opinion about the function of the stomach, so far as that is in any way possible with our present knowledge. All other methods which have been suggested for an examination of the gastric digestion may be set aside; nevertheless, we shall mention some of them later on. In addition, we remark here that even an exact determination of the duration of digestion after a test-meal is usually unnecessary, so that the examiner can generally be content with the introduction of the sound into the fasting stomach at the supposed height of digestion of the test-meal. In certain cases the stomach is found already empty at the time when the height of digestion should be expected. That would indicate an acceleration of the evacuation of the stomach, and that the duration was already at the limit of normal digestion. The next time the sound would be introduced an hour earlier.

Method of Abstracting and Examining the Stomach-contents.—*Contraindications* to the use of the sound are—a considerable hemorrhage from the stomach a short time previous; a slight loss of blood does not forbid its use if the stomach be full, but it must not be employed with the organ empty. Great general debility by diseases of the stomach ought not to prevent its use, but it is sometimes better to strengthen the patient first by artificial alimentation. As regards diseases of the heart and blood-vessels and affections of the lungs no

rules can be given here, more especially as the danger is frequently influenced by the psychical behavior (excitement, anxiety) of the patient during the use of the sound. Aneurysms without exception contraindicate the introduction of the sound.

Soft stomach-tubes must exclusively be used, and the best are the English patent sounds of Jaques, Nos. 20 to 22. They are of excellent material, but have the disadvantage that the eyes are too sharp-edged and often too small. It is necessary, therefore, to have the eyes made larger and burnt smoothly before using them. The use of a mandrin in introducing the sound is decidedly objectionable.

It is well to put over the upper end of the sound a so-called biting ring of hard rubber, which the patient holds fast with his teeth as soon as the sound is in place; but the ring must fit the sound closely.

It is very much to be recommended to ascertain on the respective patients before the first introduction of the sound how far it must be introduced to fully reach the stomach, but also not any farther than necessary. Mark the spinal process of the ninth dorsal vertebra; place the sound with its upper eye on the marked point and measure along the back and past the side of the head to the line of the incisors, marking the place of the latter on the sound by the dermatograph. If the sound is then introduced up to the mark, it is certain to have reached the lumen of the stomach if the cardia be not located abnormally deep. If it is, a corresponding portion must be added by changing the mark.

We always moisten the sound, at least at the first introduction, with a little glycerin. Others do not do so, but certainly it is somewhat preferable, and never does any harm. In children it is particularly adapted to make the sound more acceptable.

The sound may be introduced in either of two ways: Make the patient raise his head a little; then the operator grasps the sound with the fingers of the right hand as one holds a pen; put the index and middle fingers upon the tongue, and pass the sound under them till the end of the sound reaches nearly to the end of the tongue; here press the sound down a little, and then push it on moderately rapidly and draw back the hand slowly, asking the patient at the same time to swallow. Or the sound is pushed without the aid of the left hand, but in this case, in the first place, the patient must raise his head very much until the point of the sound passes over the root of the tongue. In this instant the patient brings his head to the normal position and at the same time swallows. There is resistance at the cricoid cartilage or from spasm of the glottis, both of which can always be easily overcome. If the patient breathes badly, ask him to say *ah*. This diverts his attention, and is particularly of value if, as extremely rarely happens, the point of the sound has entered the larynx and rests there.

The operator must not remove his hand from the sound after it has been introduced as far as the mark, nor must he allow the patient to hold it unless perfectly quiet and steady. It is best for patients who introduce the sound themselves to fasten it to their hand with a string, lest, as has sometimes happened, it slip into the stomach.

Great excitement, disturbed breathing, or violent vomiting past

the side of the sound sometimes obliges the operator to abandon the operation.

When the sound has reached the stomach the stomach-contents sometimes shoot out immediately in a jet, for which one must always be prepared. For this reason, at the last moment of the introduction the upper end of it is directed into a vessel, which must not be too shallow and is to be held by an assistant. In other cases the contents of the stomach come up slowly, with and without the co-operation of the patient. The latter consists in pressing, coughing, compression of the region of the stomach with both folded hands over it, and in sitting bent over. As much of the gastric contents is removed as is necessary for the examination.

If, however, the sound does not obtain anything, the stomach may be empty or almost so, or the eyes of the sound may be stopped up. In this case the sound is to be connected with a glass funnel by means of rubber tubing, and exactly 200 grams of water at body temperature is allowed to run into the stomach by raising the funnel. It is allowed to flow till all but that in the stem of the funnel has run in, and then the water is siphoned out by lowering the funnel into a vessel standing on the floor. If nothing comes out, the operation is repeated with 500 grams.[1]

By connecting the rubber tube with a Woulf's bottle it is possible, after the process of Hoppe-Seyler, to obtain isolated gases that may be in the stomach.[2] As yet, the determination of the stomach gases scarcely has a diagnostic value of its own. They consist either of the swallowed air or of gases which are formed by fermentation and putrefaction, consisting particularly of carbonic acid, hydrocarbonates, or sulphuretted hydrogen; in the latter case the gastric contents show signs of fermentation or putrefaction—in the former the result is negative.

The extraction of gastric contents for examination is frequently followed by therapeutic evacuation and washing out, with which proceedings we do not concern ourselves further here.

If the use of the sound in the early morning gives remains of food, a considerable derangement of gastric digestion exists (insufficiency in the second degree—Boas; simply motor insufficiency—Kussmaul and Fleiner); there is also dilatation. If no food-remains are found—*i. e.* if the liquid introduced comes away clear or is only made turbid by fine uncharacteristic flocks, and is not acid—the stomach may be considered empty, and there is no derangement at all or only to a slight degree (insufficiency in the first degree, atony of the stomach, according to Kussmaul and Fleiner). If considerable quantities of thick mucus appear, it signifies catarrh of the stomach. Finally, if with or without washing with water at exactly the temperature of the body there is an acid, more or less turbid liquid which gives the reaction of free hydrochloric acid,[3] we have to do with continuous secretion of gastric juice—*hypersecretion*. In this case it is well to repeat the experiment on another morning as quickly and carefully as

[1] For the rest compare Examinations of the Stomach-contents, p. 315.

[2] A fuller description is given by Hoppe-Seyler in *Deutsch. Arch. f. klin. Medicin*, Bd. 50.

[3] See below.

possible, to make certain that the secretion has not been produced by the introduction of the sound.

The experiment of *introducing the sound after a test-meal* gives more exact knowledge of the duration of gastric secretion: seven hours after Leube's test-meal, five to six hours after Kussmaul-Fleiner's, and two and a half after Ewald's, we find the stomach empty if it is healthy. It is to be noted that during menstruation digestion is generally slower, and therefore we must never make the experiment during menstruation.

If distinct food-remains are found after the periods given above, if the stomach has been found empty in the morning, at most there exists atony or insufficiency in the first degree. If no food-remains are found, but thick mucus or gastric juice containing hydrochloric acid, there is catarrh of the stomach or hypersecretion.

If food-remains are found, they must be examined further.

In order that the use of the sound may coincide with the height of digestion of a test-meal, it must be used about four hours after Leube's meal, three hours after Kussmaul-Fleiner's, and one hour after Ewald's.

If the stomach should then also be empty, it shows that there is a great acceleration of the evacuation of the stomach. The portion of food found almost always has to be examined further.

Examination of the Stomach-contents.—We collect here the principal results of the examination of the fasting stomach and of the gastric digestion after the use of test-meals. Some peculiarities, found in the examination of the fasting stomach, we add at the end.

The gastric contents, received into a glass vessel, we examine first *with the naked eye* and *then with the microscope*. We note the odor, reaction, and after this make the *chemical analysis*.

Examination by the Eye.—In the first place, determine whether there may be an admixture of bile, pus, or blood. If bile is found in a full stomach, it is always pathological; but bile can be recognized by the naked eye only when the stomach-contents are thin and but slightly turbid. If there is suspicion of pus (very rare) or blood, the respective parts of the stomach-contents are to be examined with the microscope and with the hemin test.

As for the remains of food: normally the meat, at least most of it, should be reduced to very fine particles. If the examination is made at the height of digestion of the different test-meals, with Ewald's and Leube's the roll appears as a fine delicate purée; the mashed potatoes are no longer recognizable. An equal distribution of small and easily recognizable pieces of meat indicates deficiency of acid gastric juice, while single large fragments of meat, with partly well-digested meat, shows defective mastication. As is well known, such lumps may disturb the expression from the stomach. Distinctly perceptible crumbs of roll or of mashed potatoes, while the meat has entirely disappeared, are a sign of hydrochloric-acid hyperacidity. In this case the stomach-contents consist of a remarkably thin liquid. In severe disturbances of transportation the stomach-contents are discolored, contain both pieces of roll and meat, and moreover generally bubbles of gas, which collect as foam on the surface, and the contents, instead of having the normal odor of vomited material, smell rancid-sourish

(organic acid, particularly butyric, also acetic acid); in alkaline fermentation they may smell putrid, and in rare cases like sulphuretted hydrogen gas.

Examination by the Microscope.—At the height of normal digestion the microscope reveals still defibrinated remains of meat, with the transverse striæ partly preserved; the rest is mostly indistinguishable detritus and a small admixture of mucus.

Pathologically, there are found the above-mentioned tough particles of meat (hypacidity), or remains of roll, distinct grains of starch (hyperacidity, severe hypacidity, and fermentation), great quantities of mucus (catarrh). White blood-cells, according to their quantity, indicate slightly suppurating surfaces (ulcer, carcinoma) or a ruptured abscess (phlegmon). The significance of blood has been mentioned before; blood greatly altered (macroscopically, "coffee-grounds" and the like) can never be positively recognized by the microscope; it requires the hemin test. A few epithelial cells do not mean anything; on the contrary, masses of concentrically arranged epithelial cells awaken a strong suspicion of carcinoma. If possible, it is worth while to pick up these cells, harden them in formal in the bottom of a test-tube, and make sections of them by using the freezing microtome or imbedding them in celloidin. *Schizomycetes* are always found, and in the stagnating gastric contents they exist in great quantities, but neither their quantity nor their vitality is of special diagnostic significance, and also to specify them has no independent value for diagnosis. *Sprouting fungi* are sometimes seen in remarkably large quantities when there is much fermentation. *Sarcinæ*, both the large and small forms, are found almost solely in severe disturbance of transportation.

Chemical Examination.—For the chemical examination a portion of the gastric contents is triturated in a porcelain basin and then run through a wire sieve. The examination of an untriturated filtrate gives inexact results in regard to hydrochloric acid, since it exists in a more concentrated form in solid particles, particularly in small pieces of meat, than in the liquid.

The *reaction* of the filtrate is tested by dipping a slip of litmus-paper into it. It is almost always acid. The acidity may be caused by loosely combined, or by free and loosely combined, hydrochloric acid, or by free organic acids, or, finally, as is mostly the case, by hydrochloric acid and organic acids. Acid salts also give an acid reaction, but they do not cut any figure in the different test-meals. Next, the examination is directed to a *qualitative* determination of free hydrochloric acid and free organic acids. Among these the volatile ones (butyric, acetic, valerianic acids) are, as has already been mentioned, recognized by their odor, but at the same time they are more difficult to demonstrate chemically; therefore it suffices to recognize them by the sense of smell. For free hydrochloric acid and lactic acid, however, we have simple color reactions. The examination is as follows:

If litmus-paper is reddened, the test for free hydrochloric acid follows. Among the many methods given, only those with congo-red or congo-paper and phloroglucin-vanillin deserve to be mentioned in detail.

For the *test with congo-red* we are indebted to Dr. Hübner of Mann-

heim. It is best to use congo-paper which has been saturated with an exactly 1 : 1000 solution of congo-red. If this paper is dyed a clear sky-blue by a drop of the filtrate, there is abundant free hydrochloric acid present. An indistinct blue, a blue-black, or a violet color develops if there are present, besides a little free acid, abundant acids of the fatty-acid series, particularly lactic acid, or if these alone are present and hydrochloric acid is absent. The test, therefore, has only a qualified value; even when only free hydrochloric acid is present it is not very distinct.

The *phloroglucin-vanillin reaction* is safer and more distinct. The reagent consists of phloroglucin 2.0, vanillin 1.0, absolute alcohol 30.0. One to two drops are placed on a little china porcelain plate or small spoon; mix with it an equal amount of the filtrate; distribute the liquid and heat slowly. When drying, if free hydrochloric acid is present, there appears a bright-red color; or if there is but little free hydrochloric acid, a rose-red color. If there is no free hydrochloric acid present, the drying liquid remains brown throughout. The reaction takes place even with only 0.05 per thousand of hydrochloric acid, and is therefore a very sensitive test. Large quantities of salts prevent the reaction, but these are not present in test-meals. By organic acids, even in the highest concentration, the reaction does not take place. It would only appear in the rare case of the presence of sulphuretted hydrogen (thus, for instance, in marked putrefaction, or after having taken sulphurous waters or putrid eggs). On the whole, then, we may consider the test absolutely safe for free hydrochloric acid, but not also for loosely combined hydrochloric acid, and it is therefore sufficient to make this test alone.

The *tropäolin reaction* is not so certain, and in many places is not used any more. The reagent is a saturated alcoholic solution of tropäolin OO, of which a few drops with double the quantity of gastric contents are placed on a little porcelain saucer. They are distributed by agitation, and after pouring off the surplus slowly heated. Free, not loosely combined, hydrochloric acid gives a lilac-red luster even with 0.5 per thousand; but free organic acids give the same reaction, although only in a concentration of more than 0.6 per cent.—a condition which scarcely occurs in the gastric contents.

Of the numerous other tests we only mention: The reaction with methyl-violet, which is applied as follows: two reagent-glasses are half-filled with a transparent solution of methyl-violet, and to this some of the filtrate is added. Free HCl colors methyl-violet blue. The reaction is not very distinct nor is it very reliable; it can be imitated by table-salt, and it may be concealed by albuminate, peptone, etc. There are also to be named: blue ultramarine and resorcin, recently recommended by Boas (resorcin 5 parts, sugar 3 parts, dilute spirit to 100 parts).

All these color reactions indicate only free hydrochloric acid, not the loosely combined acid also. This latter, indeed, reddens litmus-paper, but does not respond to these reagents of which we have been speaking. But as the free hydrochloric acid alone is concerned in the peptonization of albuminous substances, the reactions for free acid, particularly the phloroglucin-vanillin test, indicate directly whether the gastric juice possesses the power to digest albumin or not.

The *examination for lactic acid* is conducted in the way suggested by Uffelmann: to about 100 gm. of a 2 per cent. solution of carbolic acid we add one to two drops of a solution of chlorid of iron, when the mixture becomes steel-blue. To this we add some of the stomach-fluid. If lactic acid is present, the solution is discolored and becomes yellow or yellowish-green; on the other hand, if there is only HCl, the solution becomes clear, like water. Butyric and acetic acids give it a more yellowish-red color; moreover, they are recognized by their odor, at any rate, after shaking up some of the stomach-fluid with ether and evaporating the ether. Uffelmann's test is a very delicate one, and shows 0.01 per thousand of lactic acid. Its certainty is somewhat detracted from by the fact that lactic-acid salts give the same reaction. It is more important that alcohol, sugar, and acid salts cause the solution of chlorid of iron and carbolic acid to assume a straw-yellow color. For this reason, if there is no pronounced greenish-yellow, but a straw-yellow, coloration, we must employ a more certain method: we simply agitate some of the filtrate with ether in a reagent-glass, pour off the ether, and then evaporate the residue over hot water, not a flame. We dissolve the deposit in water, and apply Uffelmann's reaction by the addition of a few drops of the reagent (Ewald).

The *quantitative examination* for hydrochloric acid has for its object either—(1) to find out the percentage of all the hydrochloric acid secreted by the stomach: this object is to a certain extent attained by determining the free and loosely combined acid which a certain definite portion of the gastric contents contains; (2) or one is content with the determination of the free hydrochloric acid which is present in a measured portion of the gastric contents.

One might be disposed to regard the former as a considerably more exact determination, the one that best represents the capability of the stomach for chemical work; but it must be considered that the ascertained quantity of free and loosely combined hydrochloric acid at best only approximates the amount of hydrochloric acid really secreted, but never equals it, because not only is that part which is firmly combined with alkalies lost, but also that which has been reabsorbed by the stomach before the introduction of the sound, and that which has been passed on into the duodenum. Therefore exactitude is out of the question even with this method.

The determination of *free* hydrochloric acid, again, has no value at all if made after an ordinary meal or after a meal not given on an empty stomach, for in such cases an unknown quantity of hydrochloric acid is combined with albuminates, etc., and, besides, sometimes all of the hydrochloric acid may enter into combination in a healthy stomach after a hearty meal of meat. If, however, a fixed test-meal is introduced into an empty stomach, similar conditions are secured for the experiment, which, with reference to the firm and loose combinations of hydrochloric acid, may be compared one with another. The same favorable conditions of experiment are perhaps (?) procured for the reabsorption of hydrochloric acid by the stomach, while, it is true, its transportation into the intestines remains entirely uncontrollable, and certainly varies much within normal limits. Here, too, therefore, we cannot speak of exactness, but experience has established upon the foundation of innumerable

experiments what may be expected from the healthy stomach after the introduction of a test-meal. But experience has also taught that the more complicated method for an exact quantitative determination of the loosely combined and of the free hydrochloric acid, and even of the firmly combined hydrochloric acid, have no considerable advantage over simpler methods which have in view principally the *free* hydrochloric acid. In practice one may be entirely satisfied with the latter methods, provided he is severely cautious to examine only after a definite test-meal introduced into the empty stomach. Here we recommend to the practitioner to always use the same test-meal, be it one or the other. We prefer, for reasons mentioned before, one containing meat to Ewald's test-breakfast.

To the qualitative examination it is well to join the quantitative determination of hydrochloric acid.

(*a*) For *the determination of the free plus the loosely combined hydrochloric acid* different methods have been elaborated—those by Sjöqvist, Hehner-Seeman, Leo, Lüttke-Martius. We cite only the first named in a modification proposed by v. Jaksch:

Method of Sjöqvist, modified after v. Jaksch[1].—The unfiltrated gastric juice is mixed with some tincture of litmus in a crucible of platinum or nickel; add carbonate of baryta, free from chlorin, till the liquid no longer appears red; then evaporate the liquid to dryness in a water-bath. Whatever remains is then burned over a free fire, kept glowing for a short time; after cooling it is repeatedly extracted with hot water, filtered; evaporate the filtrate a little in the water-bath till it is reduced to about 100 c.cm. To the liquid is to be added dilute sulphuric acid; precipitation of sulphate of baryta takes place, which is to be placed upon a close filter, free from ashes, washed with water, brought to a glow in the platinum crucible, and weighed. The calculation is made as follows: 233 parts by weight of sulphate of baryta correspond to 73 parts by weight of hydrochloric acid. The amount of free hydrochloric acid contained in 10 c.cm. of gastric juice is therefore calculated by the following formula: (*m* equals the amount of sulphate baryta in 10 c.cm. of gastric juice, *x* equals the hydrochloric acid in 10 c.cm.):

$$x = \frac{73}{233} \times 0.3133 \times m.$$

(*b*) The *determination of the free hydrochloric acid*, after Mietz, founded upon the phloroglucin-vanillin reaction, which, as is well known, is a very safe and distinct one and well adapted as an indicator. If the qualitative examination for free hydrochloric acid by means of phloroglucin-vanillin has given a positive result, sufficient normal solution of caustic soda is added to a measured quantity of gastric contents to cause the hydrochloric-acid reaction to disappear. From the amount of alkali added is calculated the amount of free hydrochloric acid contained in 10 c.cm.

Method of Procedure.—Take exactly 10 c.cm. of gastric contents which have been ground in a porcelain crucible and pressed through a

[1] See v. Jaksch's *Kl. Diag. der Inner. Krankheiten.*

wire sieve. If too thick, mix with distilled water till a thin liquid is formed. Then add from a burette sufficient of a decinormal [$\frac{1}{10}$ per cent.] solution of caustic soda to wipe out every trace of the phloroglucin reaction from every drop of the liquid. From the amount of soda solution used the amount of free hydrochloric acid is calculated as follows: 1 c.cm. of the decinormal solution of caustic soda = 0.00365 hydrochloric acid. For instance, there have been used 5 c.cm. of the soda solution, which would correspond to 5.0 × 0.00365 = 0.01825 hydrochloric acid. Hence in 10 c.cm. there were 0.018 of free hydrochloric acid in 100 c.cm.; that is, therefore, 0.10—*i. e.* there were 0.18 per cent. of free hydrochloric acid.

This determination of the free hydrochloric acid—or, if you will, of the *excess of hydrochloric acid*—is a tolerably exact and useful one. Unfortunately, it takes up much time by the necessary, continuous repetition of the time-consuming phloroglucin reaction. Fleiner has therefore suggested a simplification which can be recommended: Before titration add to the portion of the gastric contents 25 drops of phloroglucin-vanillin; mix carefully and titrate, add the mixture drop by drop upon a porcelain spoon while carefully heating it, and sharply observe the play of colors.

In like manner, by titration, if the qualitative examination has not revealed any free hydrochloric acid, one may find how much hydrochloric acid must be added before free hydrochloric acid appears. Strictly speaking, this is a determination of how much hydrochloric acid is lacking to produce a loose saturation (of the albuminates, etc.), and we call the deficient quantity *deficiency of hydrochloric acid.*

Method of Procedure.—Prepare a pure normal solution of hydrochloric acid—*i. e.* 10 c.cm. of which are exactly neutralized by 10 c.cm. of decinormal solution of caustic soda. Now to 10 c.cm. of the chyme prepared and diluted with water, as mentioned above, add normal hydrochloric acid from a burette till the phloroglucin reaction gives a positive result. The hydrochloric-acid deficiency is calculated as applied to 100 c.cm. directly from the normal hydrochloric-acid solution used: 1 c.cm. of the decinormal solution of hydrochloric acid = 0.00365 hydrochloric acid.

Here, too, the simplification suggested by Fleiner may be employed: Mix the chyme with 25 drops of phloroglucin-vanillin before the commencement of titration.

The *determination of the total amount of acid* in the chyme has little value compared with these examinations. Nevertheless, it is occasionally of interest to express in numbers the sometimes astonishingly high acid values which occur in abnormal fermentations.

Method of Procedure.—Add to 10 c.cm. of the chyme (diluted while being stirred) a few drops of a 1 per cent. alcoholic solution of phenolphthalein, which is colorless in acid reaction, but beautifully rose-red in alkaline. Add from the burette decinormal solution of caustic soda till a reddish color-tone appears. The acidity is expressed by the amount of the soda solution referred to 100 or 1000 c.cm. of gastric contents.

In the total amount of acidity organic acids as well as hydrochloric acid almost always participate. It is specially to be observed that not

only where free hydrochloric acid besides organic acids appear, but even where free hydrochloric acid is absent, hydrochloric acid generally participates in the acidity, because the hydrochloric combinations of albumin react acid.

We generally have to refrain from the direct *quantitative determination of organic acids* because of the time required and their difficulty. Indirectly, we may approximately find them by determining the total acidity in 10 c.cm. of chyme, then make an ethereal extract in the separating funnel, and again determine the acidity. The hydrochloric-acid acidity is approximately ascertained, and, by subtracting this from the total quantity of acidity, we arrive at the acidity of organic acids.

Examining the Digestion in an Incubator.—The examination of the digestive power of the gastric juice is of especial value for demonstrating pepsin. At any rate, experience shows that when there is free muriatic acid pepsin is usually present; on the other hand, when muriatic acid is absent no pepsin is present, for the reason that the mucous membrane of the stomach does not secrete pepsin itself, but secretes its zymogen, propepsin, and because muriatic acid has the exclusive, or at least the chief, power to form pepsin out of propepsin. For these reasons it may suffice, in most cases, to examine for muriatic acid alone. But the thorough examination is of the greatest value for arriving at a complete judgment.

We test the digestive power of the gastric juice upon a piece of the white of a hard-boiled egg. A piece about a centimeter square and a millimeter thick placed in a reagent-glass full of normal stomach-fluid should be dissolved in about an hour. If the solution is delayed or does not take place at all, it proves that there is a deficiency in the normal amount of pepsin only when we are able to determine that there is also a deficiency in muriatic acid. For this reason it is best to conduct the examination simultaneously in two reagent-glasses, to one of which a few drops of HCl have been added.

The coagulating effect of the gastric juice—that is to say, of the rennet-ferment—upon the casein of milk is proved by the fact that at the temperature of the body neutralized stomach-filtrate, with neutral or amphoteric milk, is coagulated: in fifteen to thirty minutes, if the rennet-ferment is present, there is coagulation of the casein. This test, it seems, can generally be omitted if it concerns nurslings, in whom it is of special interest, for it has been shown that when free HCl and pepsin are present the rennet-ferment is never absent; even in most cases of absence of both the others rennet-ferment indeed seems not to be met with, but rennet-zymogen, which requires muriatic acid in order to transform it into rennet-ferment. In order to prove the presence of rennet-zymogen in gastric juice which is deficient in HCl and rennet-ferment, we supply the deficiency by adding HCl, and then allow it to stand in an incubator for two hours, after which we apply the test for the ferment mentioned above. In atrophy of the mucous membrane of the stomach there is entire absence of rennet-zymogen, as well as of HCl and pepsin.

Of the somewhat difficult *methods of examining the products of digestion* we can here mention the two following:

1. The transformation of the starches into erythro- and achroö-

dextrin can be qualitatively followed by means of dilute Lugol's solution (iod. 1 part, iodid of potash 2 parts, aq. dest. 200 parts): it colors starch blue; erythro-dextrin, purple-red; achroödextrin remains colorless or becomes yellow. A mixture of starch and dextrin with the first drops of the iodin solution becomes colorless, but upon further addition it becomes red and then blue.

2. Peptone and propeptone in alkaline solution upon the addition of a solution of sulphate of copper give a beautiful purple color; albumin makes it a blue-violet; hence, on account of this similarity of colors, it is often extremely difficult to distinguish albumin from peptone, particularly if the stomach-fluid is turbid.

By way of an appendix we give some methods which have only a limited value for judging of disturbances of gastric digestion. The first two seek to supply a diagnostic need which indeed exists, but in our opinion is not fulfilled by this method. The effort is made to ascertain as exactly and simply as possible how fast and how completely (if completely) the stomach empties its contents into the duodenum.

1. *The Salol Method of Ewald.*—The peculiarity of salol that it splits up into salicylic acid and phenol only in the intestine, whereupon the appearance of salicylic acid in the urine is easily proved, has been employed by Ewald to determine the rapidity of the passage of food from the stomach into the intestine. Salicylic acid is recognized in the urine after the addition of chlorid of iron by the violet reaction in the urine. In order to recognize the first traces we must make the test upon an ethereal extract [of the urine. (Compare what is said later regarding the *Urine after the Administration of Medicines.*)] Ewald found that in health the first positive reaction took place a half to one hour after the salol had been taken; when the process of transportation from the stomach has been interrupted he has seen it appear later. Contrary to him, Huber and others find Ewald's method unreliable. They do not think that the time of the appearance of the reaction, but its shorter or longer duration, should be taken into account. In a healthy person, within twenty-six hours after taking a gram of salol, salicylic acid ought to appear and then to disappear. In motor insufficiency of the stomach the reaction should last longer. Hence this method has been found unreliable.

2. *Klemperer's Oil Method.*—Klemperer has attempted a method which, from a purely technical standpoint, is seemingly very exact, but is decidedly impracticable. He introduces into the empty stomach 100 grams of olive oil, and after a certain interval washes the stomach out. From healthy stomachs he found that in two hours 70 to 80 grams of the oil had been discharged into the intestine, while in cases of catarrh of the stomach about half, and in one case of atrophy a quarter, of that amount had in the same time disappeared from the stomach. This method is less objectionable, because the oil is sometimes not borne in the patient's stomach—it may even be rejected. But it is much more so because it does not represent any stimulus

which corresponds to that which a meal of any kind exercises on the stomach. It has therefore been found unreliable by those who have tried it.

The third method, given below, examines the capability of the stomach mucosa for absorption. It is not quite perfect, *per se*, and has lately lost in importance by the circumstance that the stomach, as unquestionable investigations have shown, absorbs different substances in entirely different degrees, but upon the whole it possesses little importance for the absorption of the products of digestion, and none at all for the absorption of water.

3. *Penzoldt's Method for Examining Absorption in the Stomach.*—Penzoldt gives 0.2 iodid of potassium in gelatin capsules, and then at once tests the saliva to see whether the capsule was close and free from iodid of potassium upon its outer surface. For this purpose we have the patient, moment by moment, spit upon a piece of filter-paper saturated with a solution of starch, upon which we place a trace of fuming nitric acid; the appearance of the iodid in the saliva will be recognized by the red and blue coloration of the paper. In health the iodid will make its appearance, if it has been taken upon an empty stomach or three hours after eating, in from six and a half to eleven minutes; if directly after a meal has been eaten, after twenty to forty-five minutes. In cases of dilatation, if taken upon a fasting stomach, its earliest appearance is after fifteen to thirty minutes. It also seems to be delayed in carcinoma, chronic catarrh, and in fevers.

An improvement on this method, suggested by Sahli, consists of iodid of potash and fibrin contained in a small rubber pouch; but, in our opinion and that of others, it furnishes no clearer results.

Finally, on account of its historical interest, we mention here the method given by Leube, but superseded by his experimental meal. He introduced ice-water into the empty stomach and then aspirated it, in order to obtain for examination the gastric secretion pure—that is, diluted with water.

Results of the Examination of Stomach-digestion, and their Significance.—Repetition of statements made before cannot be avoided here:

1. If the stomach is found empty in the morning, there does not exist a severe motor disturbance, but slight motor disturbances are not excluded. If a considerable amount of food-substance remains, a severe motor disturbance exists—insufficiency in the second degree, simple insufficiency, pyloric stenosis. If the stomach contains hydrochloric-acid gastric juice, there is hypersecretion. Mucus indicates gastric catarrh.

2. If at the time of the normal height of digestion of a test-meal there are strikingly few or no food-remnants at all, the evacuation of the stomach into the duodenum is accelerated: there exists an irritable condition which may have its origin within or without the stomach.

Almost always there is much chyme of acid reaction. If it contains about 0.2 per cent. of hydrochloric acid, the gastric digestion is chemically normal, and this is the case both after test-meals and after a test-breakfast. At the same time, the lactic-acid reaction may be positive; much lactic acid, however, is not found. According to Martius and

Lüttke, lactic acid is even completely absent after a test-breakfast, and Uffelmann's reaction, if found, is said to be deceptive.

Less than 0.1 per cent. of free hydrochloric acid must be considered as pathological. There then exists hypacidity (subacidity). In this condition abundant lactic acid is often found.

If free hydrochloric acid is completely absent, we speak of anacidity. However, it must be noticed that here Sjöqvist's method, which also determines the loosely combined hydrochloric acid, almost always shows that there is secreted a greater, or sometimes also a very small, quantity of hydrochloric acid. In hydrochloric-acid anacidity there exists simultaneously the most different degrees of increased fermentation, and even of putrefaction, with odor of butyric and acetic acids, formation of gases, strong reaction of lactic acid, very high total acidity. Meat is always, starch also often, slightly or not changed at all.

If we find more than 0.2 per cent. of free hydrochloric acid, it proves that there is hyperacidity.

The utilization of these results for diagnosis must also always take into account the result of the other examinations of the stomach, and often enough also the patient's general condition. Thus, for instance, normal free hydrochloric acid with moderately retarded digestion and ectasia occurring after meals speaks for simple atony or slight and benign pyloric stenosis (scar from ulcer).

Normal amount of free hydrochloric acid and normal duration of digestion with pronounced stomach complaints may not only exist in ulcer, but also in nervous dyspepsia. The deciding point is generally the manner and the time-relation of the complaints, and the finding of a circumscribed severe pain from pressure.[1]

Hypacidity exists in catarrh of the stomach, carcinomatous and, above all, non-carcinomatous, stenosis of the pylorus, and in ectasia, in carcinoma generally, in all possible cachexias and anemias, and, finally, in neuroses. In pulmonary tuberculosis, however, the percentage of hydrochloric acid differs in different cases, just as the general functional capacity of the stomach varies. In this disease it is therefore necessary in many cases to study more attentively the chemistry of gastric digestion. As regards the behavior of the stomach in other diseases of other organs, hydrochloric acid seems to be normally secreted in heart-diseases, unless there is catarrh in consequence of defective flow of blood (Einhorn). In subacute and chronic nephritis Biernacki has often found a considerable decrease of hydrochloric acid. In diabetes free hydrochloric acid is not infrequently absent permanently or for some time (Rosenstein).

Hypacidity, and even anacidity, have very different significance according to the coincident condition of the motility of the stomach. If a deficiency of hydrochloric acid is combined with stagnation, abnormal fermentations occur; if the chyme, however, not having been sufficiently prepared by hydrochloric acid and pepsin, is transported into the intestines at the right time or even earlier, before abnormal decompositions have developed, the intestines may vicariously take up the further digestion without disturbance, particularly when the food contains few micro-organisms, is tender, and well masticated.

[1] See p. 273.

However, Kast has found that if the hydrochloric acid is weakened in the stomach by an abundant supply of alkali, considerable quantities of ethyl sulphuric acid appear in the urine, from which it must be concluded that the processes of putrefaction in the intestines are increased. Kast refers this, probably correctly, to the circumstance that the antibacterial action of the gastric juice does not come into effect. But, at any rate, as has been proven by the works of v. Noorden, the *nutrition* cannot necessarily be injured, though the chemical function of the stomach be impeded, so long as its motive power is preserved.

Anacidity, first of all, always suggests carcinoma—an empirical tenet which cannot be shaken. It is possible that the carcinomatous disturbance of metabolism is the cause of the defective secretion of hydrochloric acid (Fr. Müller). In pyloric stenosis of doubtful nature, when there is a doubtful tumor at some other part of the stomach, the accompanying anacidity is of symptomatic value. On the other hand, sometimes in carcinoma there is hypacidity, and even normal free hydrochloric acid in spite of ectasia; hence in the latter case there probably may be excessive secretion of hydrochloric acid, while, on the other hand, individual cases of long standing ectasia from pyloric cicatrization may lead to anacidity (atrophy of the mucous membrane). In some cases of carcinoma with abundant secretion of hydrochloric acid there had previously existed an ulcer.

Further, anacidity has been observed, and not only an actual absence of hydrochloric acid, but also the loosely combined acid, in the so-called atrophic gastric catarrh and in amyloid degeneration of the gastric mucous membrane.

Hyperacidity exists in the majority of cases of ulcus rotundum [perforating ulcer of the stomach], also in acute, and sometimes, though seldom, in chronic, gastric catarrh. It also occurs in nervous dyspepsia and general neuroses, both constant and periodic, and as nervous dyspepsia in neurasthenia, as gastroxynsis acuta, or in migraine. Also the gastric crises of tabetic patients are sometimes accompanied by hyperacidity. Lastly, it has been observed in depressing psychoses.

In many of these cases hyperacidity is connected with hypersecretion, and occasionally in ulcer and catarrh as well as in neuroses.

Hypersecretion, both with and without increased hydrochloric acid, occurs independently in the gastric crises of tabes and in certain neuroses (hysteria, nervousness). It is sometimes also observed with gastric ulcer, also in individual cases of carcinoma, and in acute chronic catarrh.

3. Increased amount of HCl at the height of digestion, shortening of the time (normal maximum of one hour) during which lactic acid is present, are signs of superacidity. Thus the period of digestion is shortened, or normal, or sometimes even prolonged. As evidence of disturbed amylolysis we have unchanged starch during the whole period of digestion.

Superacidity is present in the majority of cases of ulcer, also in certain nervous dyspepsias (gastroxynsis, pyrosis hydrochlorica), lastly in acute and sometimes in chronic gastric catarrh. It is also observed in the forms of insanity accompanied by depression.

Sometimes in chronic dyspepsia superacidity is combined with

retarded evacuation—*i. e.* motor weakness. This may even increase till permanent dilatation takes place. Under some circumstances there then seems to occur a secondary injury to the mucosa, caused by the superacid gastric contents remaining so long in contact with it, which injury in turn causes a gradual change into subacidity.

Vomiting and the Examination of the Vomited Material.

The act of vomiting is produced by a sudden contraction of the stomach to which, in severe vomiting, is added an energetic co-operation of abdominal pressure by contraction of the diaphragm and the transverse abdominal muscles. The contraction of the stomach is restricted to the pyloric portion, the cardiac end remaining quiet: the cardia opens whilst the pylorus closes tightly. From the not infrequent presence of bile in the last portions that are vomited toward the end of a severe effort at vomiting, it seems that during the pauses of the attack of vomiting the pylorus does not entirely close.

In this connection we do not include the vomiting, or rather the expulsion, of food from dilated parts of the esophagus when there is stenosis or diverticula.[1]

Vomiting may occur in a great variety of ways and in diseases which differ greatly in character. We suppose that the so-called vomiting-center is situated in the oblongata. This may be stimulated from the periphery, chiefly through the sensory portion of the vagus, and so give rise to reflex vomiting. Moreover, it may be stimulated directly or by impressions from other portions of the brain (central vomiting).

Children generally vomit easier than adults. There are also individual differences. Clinically, we distinguish—

1. *Vomiting* occasioned by *reflex* influences *from the stomach.* It occurs not only in all diseases of the stomach, but also in irritation of the mucous membrane of the stomach by different poisons, certain emetics, etc., and also by overloading the stomach.

2. *Reflex vomiting* caused by *other abdominal organs*, as from the female sexual apparatus in menstruation, pregnancy, diseases of the sexual apparatus; from inflammation of the peritoneum; also, in renal and biliary colic, etc.

Likewise, *vomiting* may be caused by *irritation* or *tickling of the fauces.* Probably here also belongs vomiting which occurs at the end of a severe fit of coughing, as in whooping-cough and phthisis.

3. *Central Vomiting.*—It may result from various kinds of irritation of the brain: as different evident diseases of the brain, especially tumors; in the different forms of meningitis; in neuroses, particularly hysteria; and from uremia. Vomiting occurs also in the beginning of certain acute infectious diseases, as pneumonia, scarlet fever, small-pox, erysipelas [and remittent fever].

Vomiting is almost always accompanied by certain other phenomena—previous malaise, often severe sweating, quickening of the pulse; exhaustion with the feeling of relief, but also evidences of collapse. In diseases of the brain it sometimes occurs without any preliminary indisposition, even quite suddenly and unexpectedly.

[1] See Examination of the Esophagus.

As regards the *time when the vomiting begins* in diseases of the stomach, it often (not always) follows eating. Also in peritonitis vomiting is often excited by the taking of food; but here also it takes place quite independently of this. The *vomitus matutinus* of drunkards, as a rule, regularly occurs early in the morning, when the stomach is empty. Also in certain nervous dyspepsias there are apt to be attacks of vomiting when the stomach is empty. When there is severe vomiting without phenomena of stomach or other abdominal disturbances, we must take into consideration the other conditions named above—acute infectious diseases, disease of the brain, uremia, hysteria—according to the circumstances.

The frequency of vomiting is extraordinarily variable, and is of little moment in diagnosis; only it might be mentioned that in very marked dilatation of the stomach from pyloric stenosis vomiting occurs remarkably infrequently, but in most cases tolerably regularly, at intervals of several days, but then very profusely.

There may be *eructation* in all the conditions in which vomiting occurs. It is observed especially in both slight and severe diseases of the stomach of all kinds. Sour eructation and pyrosis [heartburn, waterbrash] may be dependent not only upon abnormal lactic-acid formation, but also upon superacidity (pyrosis hydrochlorica). In individual cases combustible gases have been observed (marsh gas and probably also other gases). There occur with nervous persons very distressing and entirely odorless eructations.

The Vomit.—When we examine the vomit we notice *the quantity*, *the macroscopical* and *microscopical appearances*, *the odor*, and *the reaction*.

The chemical examination can probably occasionally enable us to judge of the character of the stomach-digestion. This is especially the case in those diseases which we cannot include in a methodical investigation, as inclination to hemorrhage, etc.[1] Of course we must consider the relation of the vomiting to the time of the last meal and of what the meal consisted. The points of view are to be taken from what has been said above regarding experimental digestion. Where there are macroscopical appearances of blood and coloring matter of bile we must further apply the chemical tests for these substances.

The Quantity of Vomit.—Here we must consider the time and frequency of the vomiting, as well as the amount of food taken. When there is vomiting from an empty stomach, usually there is only a little mucus, seldom much mucus or saliva that has been swallowed (*vomitus matutinus potatorum*), or more or less pure gastric juice (*hypersecretion*). In acute infectious diseases, diseases of the brain, uremia, sometimes scarcely anything at all is vomited.

A vomiting which seems to result from the ingestion of food, but the amount of which considerably exceeds the quantity of food and drink last taken, is an almost mathematically sure proof of dilatation of the stomach. Here the contents of the stomach may accumulate for a number of days, and then be thrown off *en masse* to the amount of several liters.

The Macroscopical Appearance.—This will naturally depend very

[1] See above.

much upon the food taken. It was mentioned above, when speaking of the experiments with digestion, that under some circumstances we can form a conclusion regarding digestion by the comminution of the food. Some foods, as coffee, cocoa, red wine, huckleberries, etc., markedly color the vomit, and may sometimes give rise to mistake, if superficially examined, by causing one to think that there has been hematemesis (the laity being not infrequently thus deceived, and hence we must be very careful in accepting the anamnesis). When preparations of iron have been taken the vomit is black, but it is also sometimes black in acute lead-poisoning. Apart from the food from some prominent constituents (when the contents of the stomach are abnormal), we can make certain important distinctions in what is vomited, just as with the sputum.

Watery, watery-mucous, mucous vomit. The first and the second named may ordinarily have two very different meanings. In both cases we have a somewhat turbid fluid, resembling saliva or fluid mucus, which is vomited from a fasting stomach. It has an alkaline reaction, and usually indicates chronic gastric catarrh. The fluid consists of mucus from the mucous membrane of the stomach and of saliva that has been swallowed. In this way the frequently mentioned "water-brash" of drunkards (*vomitus matutinus potatorum*) especially manifests itself in the early morning immediately after rising. Also such vomiting occurs (rare) in *nervous dyspepsia.* If the fluid smells sour and has an acid reaction, and if it shows the muriatic-acid reaction and power of digestion, then we have gastric juice secreted by the empty stomach—*hypersecretion.* This gastric juice is often over-acid—*hypersecretion with hyperacidity* (over 0.3 per cent.). This occurs in certain kinds of nervous dyspepsia (gastroxynsis, gastroxia; also hysteria; tabes); but also in dyspepsia following healed ulcer and acute and chronic gastric catarrh. In these cases the quantity vomited may amount to several hundred grams.

Mucous vomit. A special form of watery-mucous vomit is peculiar to Asiatic cholera. In this disease there is often vomited a great quantity of alkaline, stale-smelling fluid, like rice-water (very like the stools of cholera).[1] The small flocks resembling rice are mucous flocks. It is not possible to separate mucous from watery-mucous vomit. Sometimes a great quantity of mucus is observed in chronic catarrh of the stomach.

Bilious vomit. As previously mentioned, bile may be mixed with every vomit, and this is especially apt to be the case in very severe efforts at vomiting, so that toward the end almost pure bile is ejected. The vomit looks yellowish-green or green, and smells decidedly bilious. It exhibits the reaction of the coloring matter of the bile.[2]

A *grass-green bilious vomit*, occurring with tolerable uniformity with every act of vomiting, whether violent or not, is a not unimportant peculiarity of peritonitis and of marked obstruction of the bowels.

Bloody vomit, vomiting of blood (*hematemesis*). Blood from the nose, throat, or esophagus may become mixed with the vomit in the act of vomiting. Small quantities in streaks are usually of no significance. Large hemorrhages from the esophagus, as in varices of

[1] See below.

[2] See Urine.

the lower portion of the esophagus, and in cirrhosis of the liver, usually after it has run down into the stomach, cause severe hematemesis. Also blood from the nose, and even from the lungs, may reach the stomach and be vomited.[1] We must be careful not to confound such an occurrence with hemorrhage of the stomach. In doubtful cases the anamnesis is of less value than the examination of the stomach, nose, and lungs.[2]

Small points of blood and streaks in the vomit, even if they come from the stomach, according to our experience, are moreover generally without significance: that they are from the stomach is proved by the presence, not infrequently, of bloody suffusion of the mucous membrane of the stomach at the autopsy. Streaks of blood frequently recurring, whose source the autopsy proves to be the stomach, are not at all uncommon in cirrhosis of the liver.

Bloody vomit, from *hemorrhage of the stomach*, takes place in ulcer of the stomach, carcinoma ventriculi, portal engorgement from cirrhosis of the liver, closure of the portal vein (rarely in general venous stasis), in severe lesions of the mucous membrane of the stomach by corrosive poisons, also in general hemorrhagic diathesis,[3] in yellow fever, melæna neonatorum; in the last-named cases there usually occurs simultaneous hemorrhage of the bowels. Very decided, and sometimes fatal, hematemesis is chiefly peculiar to ulcus ventriculi (also melæna). In carcinoma we notice very frequently repeated, but always moderate, hemorrhages. Moreover, in all these conditions the vomiting of blood may be entirely wanting, either because there is no escape of blood into the stomach or because the blood is not vomited.

When we suspect hemorrhage of the stomach, which is not vomited, we are to examine the stools.[4] Sometimes in ulcer of the stomach the patient becomes suddenly pale, may collapse, or may even die from a hemorrhage of the stomach, without there being any vomiting of blood. In order to observe exactly an ulceration of the stomach, it is particularly necessary to observe uninterruptedly the stools.

Pure blood is seldom vomited, unless there is a great quantity of it, or it is vomited directly after or during the hemorrhage. Moreover, it is never of so clear an arterial color as in hemorrhage of the lungs. The blood is almost always more or less changed by the gastric juice: it is very dark, black-brown, and has an acid reaction. If it has been in the stomach for some time, as is quite often the case in carcinoma with dilatation, because the hemorrhages are usually small and there are long pauses between the hemorrhages, under the influence of the acids, by the breaking-up of the red corpuscles and the hemoglobin and the appearance of hematin, it becomes coffee-brown and of the consistence of coffee-grounds. Then, if abundant, it is easy, with some experience, and provided there is sufficient quantity, to recognize it; yet it is easy to confound it with other substances, as coffee, cocoa, etc.[5] For this reason, and because here the microscope is deceptive, it is preferable in this case always to make a special test of the blood.

[1] See p. 66.

[2] See pp. 145, 146 for further particulars regarding the distinction of hemorrhage of the lungs from that of the stomach.

[3] See Cutaneous Hemorrhages.

[4] See this.

[5] See above.

Testing the Blood.—1. Very correctly, the hemin test is generally employed, because it is certain and distinct. The following is the best way to make it: Some of the coffee-grounds material is filtered; a little of this is to be evaporated in a watch-glass. Scrape off some of the dried material, mix it with a trace of finely-pulverized salt, place the dried mixture upon an object-glass, cover it with a glass cover, and allow one or two drops of glacial acetic acid to flow under the cover-glass; then the acetic acid is again evaporated very slowly, and, after it is thoroughly dry, one or two drops of distilled water are allowed to flow under to dissolve any crystals of salt that may be present. Under the microscope there can be seen crystals of hemin (hydrochlorate of hematin) in coffee-brown or reddish-brown crystals in rhombic plates (see Fig. 109), which must be considerably magnified, as the crystals are very small.



FIG. 109.—Crystals of hemin. Zeiss's apochromatic lens No. 8, eye-piece No. 8, camera lucida. Magnified about 300 diameters.

2. The following method (an adaptation to the vomit of Heller's test for blood-coloring material in the urine, which see) leads to a result more quickly: We place some of the filtered stomach-fluid in a reagent-glass with a like quantity of normal urine, make it strongly alkaline with liquor potassæ, and heat it. The urine-phosphates are precipitated, and carry with them the coloring-material of the blood, and when blood is present we have a cloudy, flocculent, reddish-brown deposit.

Vomiting of Pus.—Pus as a macroscopically visible constituent of the vomit is somewhat unusual, and is only observed in isolated cases of phlegmonous gastritis or of abscess of a neighboring organ breaking into the stomach. Probably it can then only be observed when it pours into the stomach in such quantities and so quickly that it makes the contents of the stomach alkaline, for only thus will it avoid immediate digestion. Regarding separate white corpuscles, see below.

Fecal Vomiting (*Miserere*, *Ileus*).—In this condition either there are considerable quantities vomited which do not look distinctly feculent, probably coming rather from the stomach or the upper portion of the small intestine, and the fecal addition is betrayed by its odor, or there are distinctly fecal masses, even balls of excrement. This kind of vomit occurs in severe diffuse peritonitis and in serious occlusion of the bowels.[1] It indicates an extremely serious and, in most cases, fatal condition, yet it does not by any means have the absolutely fatal significance which was formerly ascribed to it.

As visible admixtures which can be seen with the naked eye are still to be mentioned—

[1] See Inspection and Palpation of the Abdomen.

Round worms, which come from the small intestine, probably brought into the stomach by the first efforts at vomiting, and are afterward seen in the material vomited. Their appearance is startling, but in itself has no significance. Also:

Membranous rags of echinococcus in case one should break into the stomach from the liver or spleen. In these cases the microscope sometimes shows the scolices and hooks of the parasite.[1]

Moreover, in individual cases there are found in the vomit also oxyuris, anchylostomæ, trichinæ.[2]

Microscopical Examination.—This is of very little independent value in determining the processes of digestion. In vomiting which takes place during digestion we of course expect to find portions of food in very varying conditions, according to the time the vomiting occurs.

Starch-grains in considerable quantity for the time when the amylolytic digestive period ought to be past indicate incomplete amylolysis, as is almost always produced by hyperacidity (in consequence of the too early appearance of free muriatic acid).

Mucous corpuscles are found in watery and mucous vomit; epithelium from the mouth, throat, esophagus, also sometimes from the stomach, is observed; unchanged red blood-corpuscles are very rare; in hemorrhage of the stomach the microscope generally is useless, because the red blood-corpuscles are broken up. Also, it is rare to find white blood-corpuscles that are well preserved.

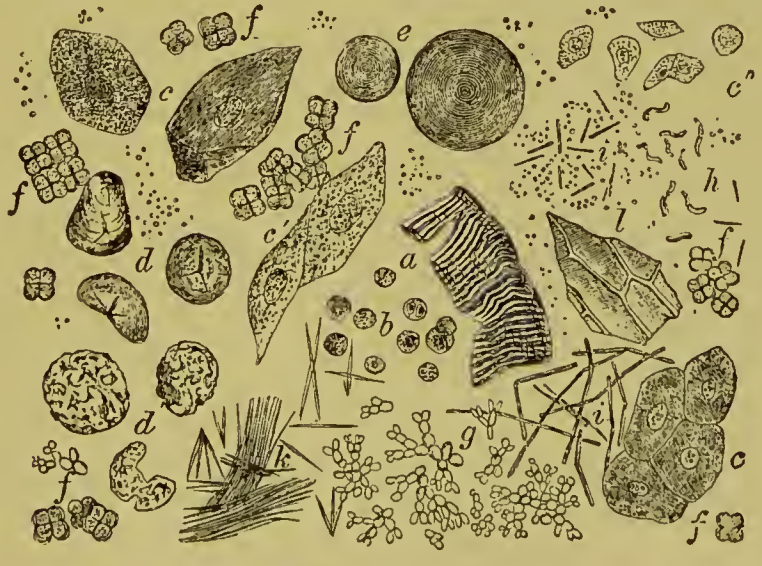


FIG. 110.—Vomited material (v. Jaksch).

a, Muscular fiber; *b*, white blood-corpuscles; *c*, *c'*, *c''*, flat and cylindrical epithelium; *d*, starch-corpuscles; *e*, fat-globules; *f*, sarcina ventriculi; *g*, yeast-ferment; *h*, *i*, cocci and bacilli (those near *h* were once found by v. Jaksch in a case of ileus, hence arising from the intestine); *k*, fat-needles, connective tissue; *l*, vegetable cells.

Sarcina ventriculi (schizomycetes) and *torula cerevisiæ* (yeast-fungus) are not entirely without value as indications that the stomach retains its contents for a long time, as, especially, in dilatation.

Of the two fungi, the sarcina is the more important. If it is not

[1] See Fig. 50, p. 158.

[2] See these under Stool.

macerated or deformed by pressure with the covering-glass it is generally easily recognized, when strongly magnified, by its peculiar resemblance to a ball wrapped with a string crossing at right angles. It is stained a reddish-brown by a weak solution of iodin or iodid of potassium.

Torulæ of different kinds and sizes (the latter very much like a small white blood-corpuscle, generally smaller) are easily distinguished as small bodies strung along together, sharply defined, which refract the light and are egg- or elliptical-shaped. Isolated ones are observed in the contents of the stomach with normal digestion. When the quantity is considerable it shows that it has been a long time in the stomach, whose contents have undergone alcoholic fermentation.

Other different kinds of bacilli and cocci, which have only recently been carefully studied, are found in the vomit, but as yet they have no diagnostic value.

Also, there are found in the vomit aphthæ (probably originating in the esophagus),[1] and favus fungus, achorion Schönleinii.

Reaction of the Vomit.—This is usually acid from muriatic or organic acids.[2] It may be alkaline when a considerable quantity of blood is vomited, as in water-brash, the watery vomit of Asiatic cholera; also, rarely, in putrid vomiting, as in ulcerating cancer of the stomach, and in the vomiting of kidney-disease.[3] Moreover, "esophagus-vomiting" manifests itself by being always alkaline.[4]

Odor of the Vomit.—In many respects this is very important. Thus, particularly, the presence of fatty acids is recognized with great certainty by their characteristic pungent odor.

The odor is very important in many poisons, as with phosphorus (odor of garlic), bitter almonds, or nitro-benzole (odor of bitter almonds), ammonia, carbolic acid, etc.

There is fecal odor with ileus, cadaveric odor in ulcerating carcinoma, also in fresh hemorrhage of the stomach.

The odor is ammoniacal in nephritic patients, especially when there is uremia. It is thought to result from the separation of urea by the mucous membrane of the stomach, by the urea in the stomach changing into carbonate of ammonia.

A penetrating aromatic odor like prunes was observed a short time ago by Eichhorst in vomited matter which contained echinococcus membranes. This same odor was noticed by him in the sputum when an echinococcus had ruptured into the air-passages.[5]

Examination of the Feces.

As in examining the contents of the stomach, the inquiring physician must pursue his task from two points of view:

On the one hand, from the character of the intestinal discharges, he is to draw a conclusion as to the intestinal digestion and any possible disturbances of it from the abnormal chemical changes, and also an opinion regarding the present disease. On the other hand, he is to form a diagnosis directly from the occurrence of certain products of

[1] See above. [2] See above, Examination of Digestion. [3] See below, under Odor.
[4] See under Examination of Esophagus. [5] Compare p. 147.

disease, or even of substances generated by disease, as intestinal parasites or micro-organisms found in the stools. Unfortunately, an explanation from the point of view first mentioned is difficult for several reasons: first, because we have to do with the last step of an extremely complicated process, and then in many respects we do not sufficiently understand this process itself or its pathological variations. With reference to the other point, and especially regarding organic exciting causes of disease, we have only a few sure principles, part of which are old and part have only recently been acquired.

We have to consider—

The *intestinal discharges*, with reference to their frequency and their possible, usually subjective, accompanying symptoms.

The more particular examination of the *stools:* quantity, consistence, or form, color, odor. In addition, there are the admixtures which are visible by the naked eye and those to be seen only by the aid of the microscope.

As yet, in our opinion, it is not possible to form an unimpeachable estimate of the intestinal digestion by the character of the intestinal secretion. It is well known that sometimes (especially by evacuating the fasting stomach) there enters into the stomach a fluid mixed with bile which is to be regarded as a mixture of pancreatic and intestinal fluids, since with an alkaline reaction it digests albumin, starch is changed into dextrin and maltose, and fat is split up. But this occasional occurrence has been very little employed for consecutive examinations. Boas recently, in a series of cases of habitual vomiting, by means of a stomach-tube, succeeded in obtaining a juice which he, as it seems to us, correctly regards as intestinal juice. However, we have not been able to convince ourselves that the results which this author has had in his clinical experimental examinations furnish a distinct picture of the processes in the uppermost part of the small intestines. They have, therefore, as yet no value for the purposes of diagnosis.

Intestinal Discharges.—In health their frequency varies individually very much. Ordinarily, at all ages, excepting nursing children, who have three or four movements a day, there is one stool in twenty-four hours, but many persons regularly have a movement twice in the twenty-four hours, while others only have one in two or three days, or even at longer intervals, without experiencing any inconvenience [or disorder]. But in scarcely any other way do physiology and pathology so much encroach upon each other's limits as with reference to the frequency of the intestinal discharges, for sometimes a movement even once in two days may be troublesome, and the physiological habitual constipation in many cases cannot in any way be distinguished from the pathological condition.

Constipation, or, better, *pathological constipation*, is called *obstipation;* the expression obstruction (severe obstruction) is often intentionally used for constipation in a serious sense. The opposite to this condition is looseness, *diarrhœa.*

The frequency of the discharges is directly connected with the quantity of food taken; hence a person who is fasting is always constipated. This point must often be thought of. The character of the food, too, has an influence upon the frequency of the discharges and

upon the passage of food through the intestinal canal.[1] Thus, rapid peristalsis causes diarrhea—slow peristalsis, obstipation. Hence any mechanical obstruction in the alimentary canal brings on constipation.

Diarrhea is the most important sign of intestinal catarrh. This is brought about by errors of diet, by cold, by infectious causes, as the intestinal catarrh of typhus, dysenteric inflammation of the large intestine, and also many intestinal catarrhs which were formerly referred to the cause first mentioned. In this condition the stools are always thin;[2] their frequency may be increased, even to occurring hourly or yet oftener.

Moreover, medicines or poisons may increase the peristalsis alone or intestinal catarrh, and thus result in diarrhea.

In all these cases the increased peristalsis increases the fluidity of the intestinal contents, even causing effusion from the intestinal wall into the intestinal cavity (cholera), until we have the condition of diarrhea, regarding which, see below.

Obstipation may be a disease which is relatively harmless, although very troublesome: *habitual obstipation*. But it is of much greater diagnostic significance, however, as an early sign of peritonitis from paralysis of the intestine. Of still greater importance is *severe obstruction* in all forms of stenosis of the intestine, as fecal accumulation, particularly in the cecum; strangulation, invagination, intussusception of the intestine; new formations, scars in the intestinal wall, compressing tumors external to the intestine; constrictions, bends produced by peritoneal exudations. In many cases of chronic intestinal occlusion, as in chronic peritonitis, constipation alternates with diarrhea.

But the condition of obstipation or diarrhea is still more affected by a possible increased or diminished abstraction of fluid from the intestinal contents: the more fluid there is, the quicker it passes through the bowel. Now, if the intestinal contents, as a result of prolonged retention, part with much fluid when there is slow peristalsis, they become dry and hard, hence are carried forward with difficulty. If the peristalsis is quicker, the contrary exists. The effect of slow or quick peristalsis is felt in the transit [of the intestinal contents], causing either obstipation or diarrhea. This is the explanation of the fact that usually obstipation and firm stools, and diarrhea and loose stools, coexist.

The severest diarrhea occurs in *cholera Asiatica*, because in this disease there is great effusion of fluid from the intestinal wall into the lumen of the intestine.

1. It is to be understood that an ordinary constipation and severe obstruction are to be sharply distinguished from each other, for a quite ordinary obstipation may be very obstinate. Here the decision is made by considering other phenomena, as vomiting, pain, and particularly by examining the abdomen. This can never be omitted in any sudden attack of obstipation, special attention being given to the hernial orifices and the cecum.

2. Persons who eat little or nothing, whom many things either strangle (stenosis of the œsophagus) or cause vomiting, as in diseases of the stomach, but especially pyloric stenosis, in which case there is infrequent but copious vomiting at a time, cannot have frequent

[1] See under Quantity. [2] See the second section below, and Consistence of the Stools.

stools; hence they must be obstipated. Such cases are easily overlooked, particularly if the patients complain a good deal of obstipation.

The special peculiarities which precede the examination of the bowels are of diagnostic importance:

Pain with the Stools.—There will be pain at the anus or at the lower portion of the abdomen in all kinds of inflammatory affections of the anus, the rectum, or their neighborhood. We have severest pain when the lower portion of the rectum is compressed by a large inflammatory (purulent) exudation, especially in the exudation of peri- and parametritis; also in fissure of the anus and abscesses from periproctitis.[1] Likewise in carcinomatous, syphilitic, gonorrheal stenosis of the rectum, but also in the usually harmless *hemorrhoids*, the pain at stool is characteristic. Sometimes in all these conditions, and particularly in all inflammations of the large intestine, but most pronounced in dysentery, there is usually painful straining at stool, and pain after it—*tenesmus*. Whenever there is pain at stool there must be a careful inspection of the anus and palpation of the rectum.

Involuntary discharges of the bowels, *incontinentia alvi*, are most frequently dependent upon the cloudiness of intelligence which accompanies any severe disease; but they may result from paralysis, particularly in diseases of the spinal cord. When the stools are thin, incontinence occurs with less loss of intelligence than if they are firm. Slight incontinence manifests itself sometimes by the fact that the patient must hasten to go to stool as soon as he has the impulse. Incontinentia is opposed to *retentio alvi* as regards its neurotic origin.[2]

Physical and Chemical Peculiarities of the Feces.—Amount.—Assuming an unobstructed passage, the amount of the stools is determined by the quantity and quality of the food taken. In the latter respect it depends upon how much of the food is digested and taken up; hence all vegetable foods make copious stools.

Also, the quantity of the stools is increased in diarrhea, because too little of the fluid portion of the intestinal contents is taken up. The greatest increase occurs in cholera from the effusion of quantities of fluid into the intestine.

Enormous quantities of firm, solid stools may be passed after prolonged obstipation or serious obstruction.

We may form an estimate of the resorption of food from the amount of the stools or of their weight if we know how much of resorbable substances the food taken contains, and if we can decide that a particular stool comes from the food taken within the period of observation, by the admixture of substances which give a distinctive color. However, we neglect the addition made to the feces during digestion from the digestive juices: on the one side, there is a too rapid movement of the food along the alimentary canal, and, on the other, disturbance of the resorption of the food. We learn from the recent investigations of F. Müller and Abelmann that in mild enteritis and in mild amyloid degeneration only the fat, but in severe cases of disease of the mucous membrane all the nutritive material, is poorly resorbed; further, that if there is a deficiency of pancreatic juice fats are split up, but (with the exception of milk) are not absorbed, and only about half of

[1] See Surgery.

[2] See Examination of the Nervous System.

the albuminous substances are absorbed; deficiency of bile and tuberculosis of the lymphatic glands disturb the absorption of fat; finally, that absorption is only slightly disturbed by accumulation in the intestinal canal.

Consistence or Form of the Stool.—Normally, it is firm or mushy. The fact has already been stated, and the reason given, why in diarrhea the stool is more or less thin or like thin soup. The stool may really be watery, as in cholera Asiatica, but also in all severe acute cases of enteritis, also in dysentery. The dried fecal balls which are passed with or after obstipation are very hard.

The *form* of firm feces does not have any independent value. Especially the stool, which is like the stool of sheep (small, hard balls about the size of a cherry), is not characteristic of stenosis of the rectum, because it also occurs in ordinary constipation. Band-like flat scybala rather indicate stenosis, more especially compression of the rectum antero-posteriorly.

Here may be mentioned the arrangement in layers of the thin and the mushy stools which not infrequently are met with. In these the firm portions settle so that the upper part of the stool consists of a clear watery layer. This is the kind of stool we have in typhus abdominalis [typhoid fever], but we also have it in other thin stools, and it is very commonly a result of the admixture of urine.

Odor of the Stools.—The variations from the normal fecal odor not infrequently have distinct diagnostic value. In nursing children a slightly sour odor is normal.

The alcoholic stool is offensive, but does not always really have a foul odor. An odor like fatty acids (and acid reaction from acid fermentation) is peculiar to the slight forms of infantile diarrhea. A decidedly foul smell (putrid albumin, "alkaline fermentation") belongs to severe forms of this disease. The stools of cholera and dysentery often smell flat, like semen (cadaverin, Brieger). Cadaverous, foul, stinking stools characterize gangrenous dysentery, carcinomatous or syphilitic ulceration of the rectum. When blood or pus is mixed with the stool in considerable quantities, the fecal odor may be masked and replaced by a mild, stale odor. Often the stool is ammoniacal from admixture with urine which has decomposed.

Reaction of the Stools.—Only in children, particularly nurslings (in whom it is normally slightly acid), is the reaction diagnostic and gives important indications for treatment. Decidedly acid reaction is observed in acid fermentation in the intestinal canal; alkaline reaction in alkaline fermentation, with putrid albumin. In both conditions there is intestinal catarrh.

Color, Constituents, Admixtures of the Stools (so far as they can be recognized by the naked eye).—The normal color of the stools varies from bright- to blackish-brown. It is in part due to the addition of bile (that is, products of decomposition of the coloring matter of the bile, particularly hydrobilirubin) and partly to the food. By the latter the stool may be unusually colored, as by huckleberries, which color it black, and may be confounded with blood.

In the normal stool portions of food can be recognized with the naked eye if things that cannot be digested—like cherry-stones, par-

ticles of wood, etc.—have been swallowed. We also see grape-seeds, the skin of many kinds of fruits, etc. Large fibers of connective tissue, undigested portions of grains, mushrooms, etc., may sometimes be met with in the stools if the patient has eaten rapidly or has swallowed his food in quantities. With the naked eye we can see fibers and pieces of undigested substances (the old designation for which was *lientery*), like portions of muscle, flocks of casein, in the stools of children, sometimes somewhat friable, perhaps slimy, or even portions of starch. All of these indicate disturbance of digestion in the small intestine, or also in the stomach, as is seen in intestinal catarrh or catarrh of the stomach, or in the dyspepsia of fever, with increased peristalsis.

In the rare condition of communication between the stomach and colon (perforating ulcer of the stomach) we find the coarsest admixture of digestible portions of food in the stool.

Occasionally, extraordinary forms of remains of vegetables (orange-like, etc.) have given rise to mistake. With children, hysterical persons, and imbeciles we must be prepared for all sorts of preposterous foreign bodies in the stools.

The stools of nurslings and of adults who live upon milk illustrate the appearance of the stool when colored only by bile-pigment. Firm stools are generally darker than thin ones, because more concentrated. In severe diarrhea, but especially in cholera, dysentery, also severe enteritis, after the first evacuations have swept out the intestinal contents the stools always become brighter, afterward grayish-white and watery, or, in dysentery, colored by blood, etc.[1]

When there is *diminished flow of bile* into the intestine, as occurs in hepatogenous icterus, the stools are lighter. If the bile is cut off, they are grayish-white, clayey, and faintly glistening. This is due not alone to the want of the transformation of the bile-pigment, but also, it would seem, chiefly to the large amount of fat in the so-called *acholic stools*. The increased amount of fat, in turn, shows diminished digestion of the fat, due to the deficiency of bile.

We designate as *bilious stools* those which contain the coloring-matter of the bile unchanged. A quick passage of the contents of the intestine and profuse diarrhea always bring about this kind of stool. We see it most frequently in acute intestinal catarrh, especially in children; perhaps there is here also an increased effusion of bile. The bilious stool is bright-yellow, green-yellow, or green, and has the reaction of the coloring-matter of the bile. We filter it and treat the filtrate as we do when testing for bile in the urine.[2]

Mucous Stools.—When mucus can be distinctly recognized in the evacuations of the bowels, it always indicates catarrh of the mucous membrane of the intestine, and hence something pathological, though in many cases the disturbance in the intestines may be regarded as without significance. There are unnoticeable transitions from the normal secretion of mucus by the intestine to a decided stimulation by chemical or mechanical irritation, even to a true enteritis. Nothnagel considers that small, visible particles of mucus interspersed in firm stools belong to a normal condition.

Larger masses of mucus in the form of more or less thick shreds

[1] See below.

[2] See this.

always indicate with greater probability a catarrh of the large intestine. Certain small, roundish particles of mucus, like sago-granules, are said to come usually from this portion of the intestine. *Catarrh of the large intestine*, then, can be definitely diagnosed from the stools if firm fecal balls are passed which are covered with mucus. Sometimes we find spread over the scybala a layer of thick, tough mucus; this occurs, however, only in chronic catarrh of the large intestine. An abundant admixture of mucus in thin stools occurs, especially in acute intestinal catarrh, if the large intestine is also affected, and in catarrhal dysentery.

We designate as *intestinal infarction* cylindrical tubes which consist entirely of mucus (or partly of fibrin) and which form casts of the large intestine. In rare cases they occur in chronic catarrh of the large intestine, and are usually passed with great pain (mucous colic).

If there are fine and equal portions of mucus in solid fecal balls, we then think of *catarrh of the small intestine*. But, also, mucus occurring in thin stools may have its origin in the small intestine. Then it is usually finely divided and is soft. In cholera Asiatica (also in cholera morbus) the stools are watery and contain particles of mucus which look like boiled rice (*rice-water stools*).

Nothnagel utters a warning against regarding all small, slimy-looking particles in the stools as mucus. They may also come from the food.

Watery Stools.—To these we have already referred repeatedly. They occur in severe acute intestinal catarrh, in dysentery, and in cholera Asiatica, and express profuse diarrhea, by which the intestinal contents are completely expelled. Even bile or its transition products are not usually found in watery stools.

Fatty Stool.—This is usually recognized by its slightly glistening and its greasy look. When there is much fat, the stool is clayey-looking or whitish, even when the bile is not cut off from the intestine. When the stool contains considerable fat, moreover, it has the peculiarity of becoming softer and more glistening with the elevation of the temperature of the body. (For further regarding fatty stool and its occurrence, see under Microscopical Examination.)

Bloody Stools.—These have an extremely variable appearance, dependent upon the more or less change in the blood, and whether it is not at all or is intimately mixed with the feces.

When firm scybala are covered with blood, they indicate hemorrhage of the rectum or large intestine. If the blood does not look at all changed, it is from the rectum or anus. When there is an admixture of blood with thin stools, if the blood retains its color and is not intimately mixed with feces, mucus, or pus, it points with tolerable certainty to the large intestine or anus. However, there may be intimate mixture of blood even in hemorrhage from the large intestine and in watery stools, as in meat-juice stools in dysentery and severe catarrh of the large intestine in children.

Hemorrhage of the large intestine occurs most frequently with *hemorrhoids* in the lower portion of the rectum, carcinomatous ulceration, again chiefly from the rectum, and in other ulcerations of the large intestine of any kind, as in dysentery.

When the *blood* is intimately *mixed with the feces*, it indicates hemorrhage from the small intestine or from the stomach. Besides, in this case the blood is usually more or less changed, brownish-red, even deep-black, the color of tar, from the breaking up of the red corpuscles and of hemoglobin (formation of sulphate of iron ?).

The degree of change which the blood undergoes depends upon the length of time it has been in the intestinal canal and the way in which it is mixed with the feces. There is the least change, the blood sometimes remaining red, with preservation of the red corpuscles, when a large quantity of blood from the lower part of the ileum passes quickly into the colon because of existing diarrhea. This happens with the profuse hemorrhage of the bowels in typhus abdominalis [typhoid fever]. Blood which comes from the stomach and duodenum (in ulcer of the stomach, *ulcus duodenale*) becomes as black as tar before it is evacuated, because of its slow transit and the usual absence of diarrhea. Moreover, with gastric hemorrhage the blood in the stool may have the appearance of coffee-grounds.[1]

In most cases in order to prove the existence of blood it does not suffice merely to examine with the naked eye. Then we employ the microscope to make out the red blood-corpuscles, and if they are broken up, then it is necessary to *test for hemin*.[2]

1. We have already repeatedly spoken of the importance of giving continued attention to the stools whenever there is a suspicion of hemorrhage in the alimentary canal. This obtains particularly with ulcer of the stomach or duodenum.

2. It is evident that any blood which reaches the stomach, having its origin in the esophagus or coming from farther up and being swallowed, may appear in the stools.[3]

Purulent Stools.—A considerable quantity of pure pus is not so very rare, happening as a sign of a rupture somewhere of a collection of pus (generally of a parametric exudate) into the intestines, especially the rectum. Therefore, whenever there is a febrile affection of the abdomen, where the formation of the pus is either made out or at least is thought to be possible, we ought always, but especially if there has been a sudden decline of the fever, carefully examine the stools as well as the urine.[4] Moreover, dysenteric, catarrhal, syphilitic, and carcinomatous ulcerations of the large intestine produce some, or possibly considerable, accumulation of pus, according to their extent; likewise, periproctitic abscesses.

While larger and especially flocky admixtures of pus are easily detected by the naked eye, finer admixtures of pus may escape it, especially when the stools are thin and of a light-yellow color. It is therefore advisable when there is a suspicion of pus to use the microscope.

Gall-stones; Enteroliths.—The former either come from the gall-bladder or the intrahepatic gall-passages (intrahepatic stones, much smaller than the others, rare) through the ductus choledochus, and, as they come into the intestine, often produce severe colic and jaundice. Whenever there is abdominal colic, particularly if it is connected with

[1] See above, p. 329.
[2] See above, p. 330.
[3] See Examination of the Nose, Expectoration, Esophagus.
[4] See this.

jaundice, and generally whenever there is jaundice, we must look out for gall-stones in the stools. In rare cases, if there is suppuration of the gall-bladder, they come from the gall-bladder, there being adhesion with the colon, into which they break and thus directly reach the intestine.

When we are looking for gall-stones the stool must be passed through a sieve. If it is formed or mushy, it must be broken up by pouring a stream of water upon it. The gall-stones are generally very easily recognized by their shining appearance, smooth surface, and irregular, many-angled (facets) form. Small, especially intrahepatic, stones may not have facets and be more crumbling. They consist chiefly of cholesterin, and also contain coloring-matter of the bile.

The observations of Fürbringer show that when people have eaten pears, particularly those which contain many concretions, these concretions, appearing in great abundance in the stools, may be mistaken for gall-stones. They are white-yellow, loam-colored to red-brown formations, of the size of a grain of sand to that of a pea, and sometimes still larger. They give neither the reaction of cholesterin, nor, if cleansed from feces, of bilirubin, and consist microscopically of stone-cells. It is conceivable that they might give rise to an erroneous diagnosis of gall-stone colic if found in the stools of a patient with icterus or complaining of colicky pains.

Methods of Chemical Examination of Gall-stones.—(*a*) *Test for Cholesterin.*—Pulverize; dissolve in hot alcohol; filter; allow one drop of the filtrate to fall on a glass slide. When dry the characteristic plates of cholesterin are seen.[1]

(*b*) *Test for Bilirubin.*—The remains on the filter are to be slightly acidulated with hydrochloric acid, mixed with chloroform with warmth, and allowed to stand a moment. The decanted chloroform forms with nitric acid a beautiful green.

Enteroliths are rare. They usually come from the vermiform appendix, and their center commonly consists of solid, undigested portions of food, as a cherry-stone, around which have been deposited some lime or magnesium salts.

Portions of Tissue from the Intestinal Canal.—In very rare cases, when there is invagination of the intestine, the whole of the portion that is turned in sloughs off, the intestine forming new adhesions, and thus life is preserved. This entire piece may appear in the stool. Shreds of mucous membrane from the large intestine in dysentery, portions of tissue of carcinoma, or other new formations may appear in the stools.

Animal Parasites.

In what follows it will be shown that some of the animal parasites that exist in the human alimentary canal have no pathological significance; others, on the other hand, are very important factors as exciters of disease. The examination for these latter or for their eggs cannot be made too frequently or too carefully. An examination of the stools for parasites must be undertaken not alone when there are complaints of symptoms which directly indicate intestinal parasites, or in general

[1] See p. 156.

when there are evidences of intestinal catarrh, but in any case of anemia —when there is any general nervous depression—in certain other phenomena of the nervous system [1] if the cause of the particular complaint does not appear to be clear. The cases are numberless where, after long fruitless search elsewhere, the discovery of a joint of a tapeworm, for instance, leads to the correct apprehension and treatment of the patient.

In order not to separate what belongs together, we collect here all that is to be said regarding the occurrence of intestinal animal parasites and their eggs in the stools, whether in the examination we employ the naked eye, the simple or the compound microscope.

Tape-worm (Cestodes).—Its habitat is exclusively the small intestine. It gives rise to very great pathological disturbances (intestinal catarrh, anemia, nervous manifestations of varying severity). It consists of a very small head and neck and a ribbon of flat joints (proglottides) several meters long, which constantly push off at the end of the worm and grow again from above. It clings to the wall of the intestine by its head.

It can be recognized by a single joint, which can easily be seen with the naked eye, or by the presence of eggs in the stools (microscopical examination).

1. **Tænia Solium.**—This is 2 or 3 m. long. Its head is the size of the head of a pin, glistening gray; the rest of the worm is white or yellowish-white. Upon the head are four pigmented sucking-cups (to be seen with a simple microscope), which surround a crown of chitin-hooks, "crown of hooks." The ripe proglottides—that is, those on the lower end of the worm—are about 10 mm. long, 5 or 6 mm. broad, and are like gourd-seeds (but are smaller). From the peculiarity of these ripe joints, which are continuously thrown off and passed with the stool, we are able to make the differential diagnosis between this and the other



FIG. 111.—Tænia solium, head enlarged (Heller).

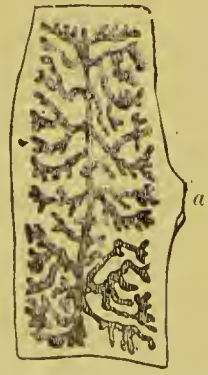


FIG. 112.—Tænia solium. Ripe joint, magnified 6 times (Heller).



FIG. 113.—Egg of tænia solium (Heller).

tape-worms. The joints show a longitudinal canal (the uterus), from which, toward both sides, as many as a dozen branches go off, which ramify like the branches of a tree.

The eggs of *T. solium* (which require the use of a moderate microscopic power in order to find them, stronger to examine them carefully)

[1] See works upon Pathology.

are round, and, if they are ripe, have very thick shells (which show radiating lines, and which, with a little pressure upon the covering glass, break into hard pieces). In the finely granular contents we often see a few chitin-hooks.

2. The **Tænia mediocanellata** seu **saginata** grows to 4 or 5 m. The head is somewhat larger than that of the solium, is also more strongly pigmented. It has no crown of hooks, but four sucking-cups, which are much stronger than those of the solium. On the whole, the rest of the worm, as respects its individual joints, is fatter and thicker than the first named. The ripe proglottides are passed not only by the stool, but wander independently from the anus, having strong, very energetic, independent movements. They are distinguished from those of the T. solium in that the uterus gives off more and finer branches on each side, which divide dichotomously.

The egg of the *T. mediocanellata* looks extremely like that of the *T. solium*, except that on the average it is somewhat larger.



FIG. 114.—Tænia mediocanellata. Head darkly pigmented (Heller).

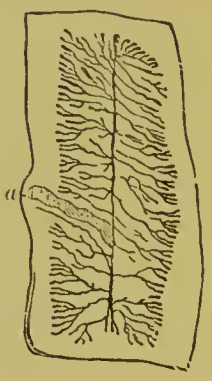


FIG. 115.—Tænia mediocanellata. Ripe joint, magnified 6 times (Heller).

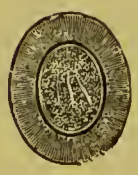


FIG. 116.—Egg of tænia mediocanellata (Heller).

3. **Bothriocephalus latus** (*sinus head*) is found in South Germany, especially in South Bavaria, in the countries bordering on the North and Baltic Seas, likewise in the neighborhood of Lake Geneva and in the Baltic Provinces of Russia [in Sweden, Poland, Belgium, and Holland. "Low-lying damp regions near the borders of seas and lakes are those in which it is most often abundant"]. It [is the largest of the tape-worms, and] attains to 7 or 8 m. in length. Its head is elongated, and has two narrow, long-drawn-out sucking-cups. The illustration (Fig. 118) shows its form and the shape of the uterus. The ripe joints are not given off singly, but a large piece of the worm is always passed at one time, and then, after a long interval, another; most frequently in the spring and fall.

For this reason we here refer to the finding of the eggs (which are always present in the stools). They are oval (see Fig. 120), and much larger than those of the two other kinds of tape-worm. The shell is bright brown, relatively thin, and on one end of the oval has an opening

which is closed with a cover of exactly the same kind. The contents of the egg are granular.

As has recently become known, the bothriocephalus gives rise to severe anemia, with changes in the blood like those in severe pernicious



FIG. 117.—Head of bothriocephalus latus, enlarged (Heller).



FIG. 118.—Ripe joint of bothriocephalus latus, enlarged 6 times (Heller).



FIG. 119.—Egg of bothriocephalus latus (Heller).



FIG. 120.—Egg of bothriocephalus latus, with developed embryo (Leuckart).

anemia; for this reason, and because there are no joints thrown off, this tape-worm is very easily overlooked for a long time.

4. **Tænia cucumerina,** 5–20 cm. long, 2 mm. wide; the head is somewhat long and has sixty hooks; the last joints are reddish and have the form of pumpkin-seed. Six to fifteen of the eggs lie together in the so-called cocoon. This worm occurs in dogs, cats, and not infrequently in man, especially children (Leuckart). Its pathological significance is not known (see Fig. 121).

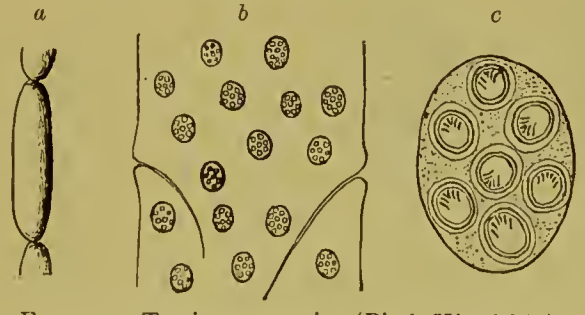


FIG. 121.—Tænia cucumerina (Birch-Hirschfeld).
a, joint, natural size; *b*, the same with cocoons, enlarged 12 times; *c*, cocoon, enlarged 290 times.

Round Worms.—Ascaris Lumbricoides.—This is easily recognized from its likeness to the common earth-worm. Its habitat is the small intestine. Very frequently it gives rise to little or no complaint, but sometimes, and especially in children, it causes very uncomfortable phenomena of all sorts, particularly of the nervous system. Occasionally, when there is severe vomiting [and sometimes when there has not been any vomiting at all], it gets into the stomach, and is then vomited. Moreover, it may crawl into the ductus choledochus and thus cause obstinate jaundice. These worms appear in the stools, and sometimes, in sleep, they will crawl out of the anus. They may sometimes come out of the mouth and nose while the person is sleeping.

The fresh eggs of the ascaris lumbricoides have a very peculiar appearance, since their chitin capsule is covered with an uneven, as it were, humped albuminous envelope (see Fig. 122).



FIG. 122.—Ascaris lumbricoides (v. Jaksch).
a, worm, natural size; *b*, head; *c*, egg.

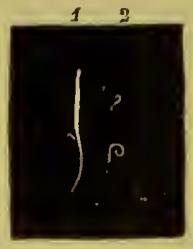


FIG. 123*a*.—Oxyuris vermicularis, natural size (Heller).
1, Female; 2, male.



FIG. 123*b*.—Egg of oxyuris vermicularis, enlarged (Heller).



FIG. 123*c*.—Oxyuris vermicularis, enlarged (Heller).
a, ripe but unimpregnated female; *b*, male; *c*, female containing eggs.

Oxyuris vermicularis is a small, white worm (Fig. 123*a*) found particularly in the large intestine. It may wander from the anus into the vagina. It has very slight pathological significance. It appears in the stools, and also it is not infrequently found by itself in the neighborhood of the anus. When first passed it has usually very lively peculiar movements. The eggs are commonly unsymmetrical (see Fig. 123*b*).

Anchylostoma duodenale is very like the last in form, but often longer, even twice as long; it usually inhabits the upper part of the small intestine, especially the duodenum.

Formerly it was only observed in other countries than Germany [discovered by Dubini in 1838 in Northern Italy], more recently also in

Switzerland (first during the building of the St. Gothard tunnel), and finally it was noticed among the inhabitants of the German mountains and among brickmakers. Because it continually sucks blood from the wall of the intestine it causes severe, sometimes fatal, anemia (*anchylostomiasis*, formerly "Egyptian chlorosis," Griesinger). It is difficult to discover the worms in the stools unless some vermifuge is used, but, on the other hand, the tolerably characteristic eggs are always present. They are as large as, or perhaps a little larger than,

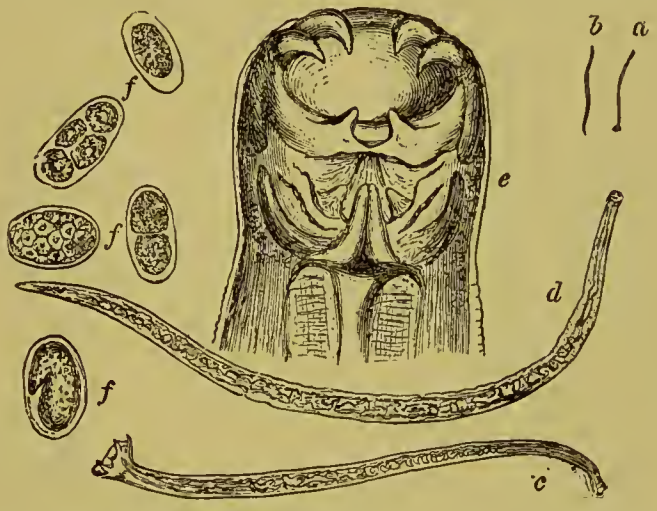


FIG. 124.—Anchylostoma duodenale (von Jaksch).
a, male; *b*, female, natural size: *c*, male; *d*, female, slightly magnified; *e*, head; *f*, eggs.

those of the oxyuris. They have a thick covering, and contain two or more segmentation globules. By allowing the stool to stand for several days in a warm place we can see the embryos develop in the eggs. In this very serious disease the stools often contain blood.

Besides the intestinal parasites already mentioned, there are the following, part of which are pathologically unimportant and others are very rare:

Trichocephalus Dispar.—Its habitat is the colon, especially the cecum. It is of no importance. Both the worms and eggs are highly characteristic in form (see Figs. 125*a* and 125*b*).



FIG. 125*a*.—Trichocephalus dispar, natural size (Heller).



FIG. 125*b*.—Egg of trichocephalus dispar, moderately enlarged (Heller).

Trichina Spiralis.—It very rarely occurs in the intestine, but sometimes in the first stage of the trichinosis, the stomach-stage with intestinal phenomena, it is found in the stools. Since the early recog-

nition of trichinosis is of the greatest importance, in a suspicious case the stool is to be examined with the greatest care, best after the administration of an aperient.

The appearance of the intestinal trichina is shown in Fig. 126. It is only one-third as long as the oxyuris, and hence cannot be seen with the naked eye.

Distoma hepaticum and **D. lanceolatum**, two rare but pathologically important parasites which inhabit the gall-passages of the



FIG. 126.—Adult intestinal trichina, human. Male, female, and two embryos, the former natural size, the latter slightly magnified (Birch-Hirschfeld).



FIG. 127.—Trichina (von Jaksch).

a, male; *b*, female intestinal trichina; *c*, muscle trichina.

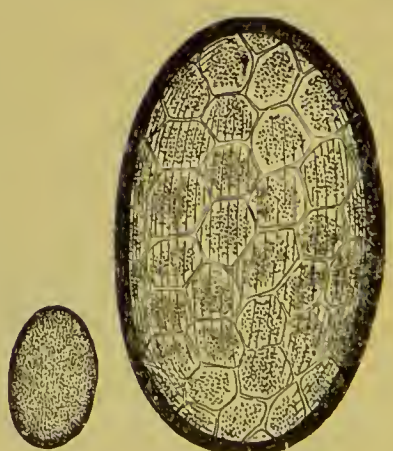


FIG. 128.—Eggs of distoma hepaticum and distoma lanceolatum, moderately magnified (Heller).

liver, sometimes make themselves known by their eggs, which, passing out into the intestine with the bile, appear in the stools. The egg of

the D. hepaticum is much larger than the other parasites previously mentioned, about three times as large as those of ascaris lumbricoides. The egg of the D. lanceolatum is somewhat smaller than that of the oxyuris. For its other characteristics see Fig. 128.

Microscopic Examination of the Feces.

Mode of Procedure.—Thin or thin-mushy stools are examined without making any addition to them. To thick, mushy, or solid stools about $\frac{1}{2}$ per cent. of solution of salt is added, and the solid portions must, of course, be broken up. Somewhat of a selection must be



FIG. 129.—Microscopical constituents of the stools (partly from v. Jaksch).
a, vegetable fragments; *b*, muscular fibers; *c*, white blood-corpuscles; *d*, saccharomyces; *e*, micro-organisms; *f*, crystals of triple phosphate; *g*, fatty-acid crystals.

made from the different portions of the stool according to the object of the examination. It has lately been recommended to mix the stools with water or a 5 per cent. solution of salt, and then to centrifuge them. The solid portions of the stool must, of course, be first broken into small particles. It is said that the stratification which results from the use of the centrifuge greatly facilitates the examination. We have not yet employed the method.

The degree of amplification is to be varied according to the object in view. In general, we employ dry lenses of high power. When looking for parasites (which have already been described) it is better, on the other hand, to make use of tolerably strong amplification.

1. Undigested Portions of Food.—These may be found in every stool, and in varying quantities according to the kind of food eaten. We mostly meet with coverings of vegetable cells, elastic fibers, etc.

2. Portions of Digested Food.—Although these, if visible with the naked eye, indicate *disturbed digestion in the small intestine*, yet microscopical particles of these substances are seen in small quantities in normal stools, as well as small portions of muscular fiber, with the transverse striations, shreds of connective tissue, starch-granules, and fat.

But considerable quantities of the substances named always indicate disturbed digestion either in the small intestine or the stomach, and hence have the same significance as the occurrence of larger pieces, which can be seen without being magnified.[1] When the microscopical particles are colored a bright-yellow, as we commonly see small por-

[1] See above, p. 347.

tions, particularly of muscular tissue, but sometimes almost all the solid portions of the stools, it shows that there is unchanged bile in the stool and catarrh of the small intestine.

Fat, in the shape of polygonal glassy lumps, of needle-shaped crystals, and also in the form of drops is a very frequent constituent of the stools. The glassy lumps occur very frequently in health, and are often colored yellow or yellowish-red. They are recognized as fat, fatty acids, or soap by their transformation upon the addition of sulphuric acid, and, when warmed, into drops of fat (Müller). Drops of fat occur in the stools with milk diet (hence, particularly in those of children), when taking cod-liver oil, likewise castor oil, and, if there is intestinal catarrh, then in very considerable amount.

The needle-shaped fatty-acid crystals are not without significance. They sometimes occur singly, and again in bundles and druses. They are changed by simply warming them, or by the addition of acid and then warming, into drops of fat, and this takes place whether they consist of fatty acids or (lime-) soap.

When there are *great numbers of fatty-acid needles* it is a pathological sign of disturbance of the resorption of fat, as may result from shutting off of the bile from the intestine, from any form of enteritis, of tuberculosis, amyloid degeneration of the intestine, and, lastly, from disease of the mesenteric glands.

However, we must mention here, further, that, according to recent investigations, after *extirpation of the pancreas*, the digestion of fat is diminished to an extraordinary degree, or even may be entirely suspended (Abelmann). In future, therefore, in cases of fatty diarrhea we must think of the pancreas.

Detritus.—With respect to detritus in the stools little needs to be said, because we cannot determine separately a great number of the kernels, husks, etc.

3. Additions to the Stools from the Alimentary Canal.—*Mucus.*—A microscopical quantity of mucus occurs in the stools of persons in health. Small glassy lumps of mucus, which come from the cells of plants, may also be present. Usually the examination with the naked eye is sufficient to determine whether there is a pathological admixture of mucus.

It is necessary only to mention that a firm stool, abundantly interspersed with small light lumps of mucus, is observed with intestinal catarrh (Nothnagel). In these cases we can generally discover the mucus, if we carefully examine, without any artificial aid.[1]

Epithelium.—Some cylindrical cells, often in mucous metamorphosis, are a frequent occurrence. If the quantity is large, it indicates intestinal catarrh. Very abundant cylindrical epithelium occurs in chronic catarrh of the large intestine, especially in mucous colic, in this case caused by mucous "infarction." It has already been mentioned[2] that regular shreds of mucous membrane are found in the stools, also portions of tissue.

Red and White Blood-corpuscles.—These are present in quantities in fresh bloody and in purulent stools. When seen but once they do not have significance.

[1] See p. 337.

[2] See p. 340.

4. Crystals.—Except the fat crystals mentioned above, there are almost no crystals which are brought into requisition for the purposes of diagnosis. Crystals of ammoniaco-magnesian phosphates[1] no doubt occur in the stools in enteritis and abdominal typhus [typhoid fever]. But they may also be found in any other stools if they are not kept separate from the urine and stand for a long time.

Lime-salts of all kinds, partly with inorganic, partly with organic, acids in the form of wedges, dumb-bells, needles, etc., sometimes colored an intense yellow by the bile in the stool, have no diagnostic import.

Charcot's crystals, in appearance and probably also chemically entirely agreeing with the Charcot-Leyden crystals of asthma, are observed in rare cases of dysentery, typhus abdominalis [typhoid fever], intestinal tuberculosis, anchylostomiasis.

5. Vegetable Parasites.—We may divide the large number of vegetable micro-organisms which we find in the stools, from the standpoint of clinical diagnosis, into two classes:

(*a*) Those which, primarily, for clinical diagnosis are only of subordinate significance, because we do not know that they have any definite connection with any diseases. Here also we class those which are indirectly harmful—that is, they cause abnormal decomposition of the intestinal contents. This class is extremely numerous, and great numbers of one kind or another are present in every stool. The knowledge of the different kinds has recently been greatly extended by the important labors of Nothnagel, Bienstock, Escherich, and others. But the point has not yet been reached which makes them as available for clinical diagnosis as the other peculiarities of the stools. For this reason we will treat of them only very briefly here.

Of the **fungus-spores** we have (very rarely) that of thrush[2] in children who are suffering from thrush in the mouth. Yeast-fungus, and, indeed, the different kinds of tortula cervisiæ (see Fig. 129, *d*), occasionally occur in all stools, especially in the milk-stools of children. In intestinal *dyspepsia* with acid fermentation they are generally more abundant than in normal digestion. But the schizomycetes belong to the numberless micro-organisms which are seen in every microscopical preparation of the stools, whether normal or pathological. Of chief importance are the micrococci and bacilli. A very large part of these are colored yellow or brownish with iodin and iodid of potassium; others are colored by the same reagent blue or violet (Nothnagel). These latter, according to v. Jaksch, are increased in intestinal catarrh.

We are now able to conclude that the knowledge of these intestinal bacteria furnish diagnostic indications of anomalies in intestinal digestion, and that the different kinds of bacilli possess extraordinary biological peculiarities. Some require for their rapid development a neutral or slightly alkaline reaction, while others an acid reaction of the intestinal contents; some are aërobiotic, others anaërobiotic; and, while some have the power to transform starch into sugar, others cause the decomposition of albumin.

(*b*) **Pathogenic Fungi.**—These we are able to isolate, and from

[1] See these under Examination of Urine.

[2] See p. 257.

them diagnosticate the disease they cause, as the tubercle bacillus in the sputum.

Here also belong the *pathogenic schizomycetes.* These are—Koch's cholera bacillus, the bacilli of abdominal typhus, and tubercle.

Cholera Bacilli (common bacilli) are the pathognomonic sign of Asiatic cholera. They are short, more or less crooked, which are sometimes connected one to another in such a way as to form "spirals" like a screw. The curve may be very slight, even wanting, or marked, even semicircular. In general, they are shorter, but thicker, than the bacilli of tubercle.

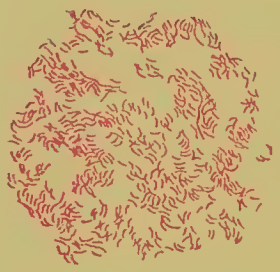


FIG. 130.—Comma bacillus, pure culture (prepared by Prof. Gärtner). Zeiss's immersion lens $\frac{1}{12}$, eye-piece No. 2, camera lucida. Magnified about 600 times.



FIG. 131.—Cholera dejections upon a damp sheet, two days old (Koch).

a, S-form bacillus, 600: 1.

Habitat; Mode of Preparation.—They are particularly found in the free mucous floccules of rice-water stools, also very abundantly upon the linen soiled by the dejections, and, indeed, here after two or three days, provided the linen has been kept moist. A mucous floccule (or a drop of the stools) or some of the deposit on the linen is placed upon a covering-glass. First dry it in the air, then pass it two or three times through the flame of a spirit-lamp, and stain it with methylene-blue or fuchsin by warming it one to five minutes. The cholera bacilli are not stained when treated by Gram's method.

These bacilli have been found, we may say, constantly in the stools of Asiatic cholera by a great many other examiners besides Koch, and they are found in no other stools. They must, therefore, diagnostically be of pathognomonic value to even those who doubt Koch's teachings concerning their pathogenic character.

But since the morphological peculiarities of the cholera bacillus in the microscopical preparation do not furnish an absolutely certain recognition, and, on the other hand, since there is no specific reaction (as with the tubercle bacillus), in order to determine an isolated or first case it is indispensably necessary to establish a pure culture.[1] It is very important to point out that even the pure culture only has decisive value when the culture medium has exactly the right degree of alkalescence.

[1] See, regarding this, the works upon Bacteriology.

Comma bacilli are also, in individual cases, found in the vomit of Asiatic cholera.

A bacillus which is morphologically like the comma bacillus occurs in tooth-mucus (Lewis and Miller), and just such an one also in old

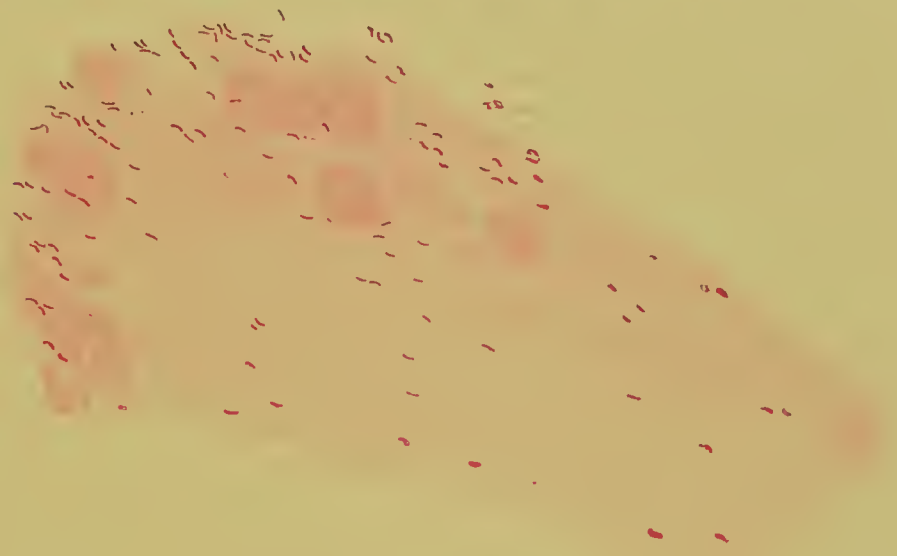


FIG. 132.—Cover-glass preparation of a mucous floccule in Asiatic cholera. Zeiss's homogeneous immersion $\frac{1}{12}$, eye-piece No. 2, drawn by a camera lucida. Magnified about 650 diameters.

cheese (cheese-spirals, Deneke). Biologically they differ from Koch's comma bacillus and from each other.

Bacilli of Typhoid Fever.—These bacilli, which have been regularly found in typhus abdominalis, in the diseased portion of intestine, in the mesenteric glands, the spleen, and liver, in the kidneys, and also frequently in the blood,[1] are, without question, to be regarded as the



FIG. 133.—Spirillum (Finkler and Prior), 700: 1 (Flügge).



FIG. 134.—Typhus abdominalis bacillus in pure culture. Zeiss's homogeneous immersion lens $\frac{1}{12}$, eye-piece No. 2, drawn with camera lucida. Magnified about 650 times.

exciting cause of this disease. They have also frequently been found in the stools and urine of typhoid fever patients. But since they are neither distinguished by their form (just at the end they are rounded; are about as long as the tubercle bacillus, but are much thicker—about one-third as thick as long) nor by a specific color-reaction from the

[1] See Appendix, and in works on Bacteriology.

other bacilli which occur in the stools, especially from the *bacterium coli commune*, which is constantly present in the intestines, their microscopical proof is wholly uncertain. But also pure cultures from the stools (and urine) usually do not give a decisive result, because they cannot be positively distinguished from the bacterium coli commune, the constant inhabitant of the intestines. Bacterial diagnosis of typhoid fever from the stools and urine is, therefore, impossible with our present resources. But the examination of the spleen-pulp or a roseola or of blood from a vein not infrequently furnishes pure cultures of typhoid bacilli which can be interpreted as nothing else, and is then decisive for the diagnosis of typhoid fever, particularly against miliary tuberculosis and septic diseases. [The method of diagnosis by serum reaction is the most reliable at present known. See page 247.]

The typhoid fever bacillus is best stained with methylene-blue or fuchsin in a dry preparation upon a cover-glass.[1]

Bacteria which are Found in Different Forms of Acute Intestinal Catarrh.—1. *Bacterium Coli Commune.*—This is found in "infectious intestinal catarrh," frequently in very great quantities. Sometimes in cholera-like rice-water stools it occurs in as great quantities and as predominantly as the comma bacillus occurs in cholera Asiatica. Often it may then lie in heaps upon and in flocks of mucus just like the comma bacillus. It may also migrate from the intestines into the various organs of the body.

It can scarcely be doubted that this constant and usually harmless inhabitant of the intestines in such cases must be considered as the cause of the intestinal disease. I must doubt whether it is much more virulent for animals if a culture be made from the stool of a case of enteritis than from a normal dejection. Besides, it shows uncommonly great differences as regards morphological conduct, chemical effect, virulence, and energy of growth. The question whether the bacillus of typhoid fever is a variety of the bacterium coli commune is generally answered in the negative by German and in the affirmative by French bacteriologists. For the present, therefore, the question must be considered as undecided.

2. *Bacterium Finkler-Prior.*—This is a species of spirillum which may be considered as the cause of some cases of cholera nostras. It is found in great quantities in the dejections of such cases, and microscopically shows a certain resemblance to the comma bacillus, but may easily be distinguished from it by a pure culture.

3. *Other Bacilli.*—Various other bacteria besides these have to be considered, but with which we are not more accurately acquainted, and they need not be considered as specific; but they exist greatly disseminated as saprophytes. They rapidly increase, especially when the atmospheric temperature is high. They act by their toxins. Milk, as is well known, is the article of diet by which, in the easiest manner, great quantities of bacteria and toxins enter the body.

Regarding a *bacillus enteritidis*, which is probably specifically pathogenic, although rare, see Gärtner's communication.[2] Beck[3] has de-

[1] For cultures, see Appendix.

[2] *Correspondenz-Blatt f. der Thüring. Aertzverein*, 1888, ref. *Baumgarten's Jahresbericht f.* 1888.

[3] Ref. *Centralbl. f. klin. Med.*, 1892.

scribed a special streptococcus, and according to some other observations it is not impossible that this streptococcus has a more important part in cases of acute enteritis than has hitherto been supposed.

Tubercle Bacilli.—These are frequently found in tuberculous ulcers of the intestine. It is not yet sufficiently established whether they are always present, chiefly because not infrequently tubercular ulcers of the intestines do not have any symptoms, and particularly do not cause diarrhea, and so, often enough, the firm stools are not examined for bacilli. On the other hand, in phthisical patients tubercle bacilli are sometimes observed in the stools without there being any intestinal tuberculosis. They come from swallowing tuberculous sputum. The method of staining is the same as in examining the sputum.

6. Animal Parasites.—Infusoria of different kinds are found in many dejections. They are probably all inhabitants of the colon, and appear in greater quantities when there is diarrhea, particularly when the evacuations consist of mucus. Catarrhal and more severe—*i. e.* ulcerous and necrotic—diseases of the mucosa of the large intestine seem specially to favor their existence and increase. Occasionally, perhaps, an increased importation *per os*—for instance, by drinking water from standing pools—may account for them. They consist of cercomonas, trichomonas, paramecium coli, balantidium coli, etc.[1]



FIG. 135.—Monads from the feces (v. Jaksch).

a, trichomonas intestinalis; *b*, cercomonas intes.; *c*, amœba coli; *d*, paramecium coli; *e*, living monads; *f*, dead monads.

It is doubtful whether these organisms have a pathogenic significance; they are probably only saprophytic attendants of the conditions mentioned.

Mode of Examination.—The fresh dejections must be examined on a warm stage. Lutz recommends to add some fresh saliva at body-temperature. The organisms can be easily found only while they move: when cooled they immediately become quiet, and are then very difficult to be seen.

Chemical Examination of the Feces.

This has great importance, which is increasing more and more at the present time from the continual improvement of the methods of examination, and by the increased means of knowledge regarding the digestion of food in the stomach and intestines of the healthy and sick.

An examination of the *food-value of the nourishment* taken consists of the following: To exactly ascertain the quantities of food introduced into the stomach, with the percentage in it of amylacea, albumin, and fat or fatty acids. The principal difficulty here is to make out which portion of feces corresponds to a certain quantity of food introduced

[1] Compare Fig. 135.

within a certain space of time. This can be done only by separating the time of the experiment by pauses of fasting, which in turn has its difficulty in man, particularly in a patient. Assistance is derived from a coloring substance, as charcoal, given at the beginning of the experiment, better still at the beginning and end of it, which substance appears unchanged in the feces and makes its demarcation possible. A second difficulty exists in the fact that the secretions from the intestinal tract and the continual casting off of epithelia, which is probably more abundant than has hitherto been believed, add to the nitrogen and fat in the feces to a degree impossible to determine.

We cannot find room in this compendium for the methods which have to be applied in this investigation, but we refer to the works of Fr. Müller,[1] of v. Noorden,[2] and Abelmann.[3]

It is also necessary to state very briefly the results of the investigations hitherto made, since we do not as yet have at hand any conclusions which are useful for diagnosis. We only mention that, according to F. Müller, in slight enteritis only the fat is imperfectly absorbed, but that in severe disease of the intestinal mucosa (extended amyloid disease) all food-stuffs are poorly absorbed; that in passive congestion of the intestinal tract absorption is very slightly impaired; finally, that deficiency of bile, as well as tuberculosis of the mesenteric glands, interferes with the absorption of fat. Regarding the effect of shutting off of the pancreatic juice, the clinical results of Müller and the experimental ones of Abelmann are opposed to each other. According to the latter, which seem to us to be conclusive, the absence of pancreatic juice has the effect of reducing the absorption of albuminous substances to about one-half of the albumin administered; that the amylacea are somewhat less reduced; that the fat, however, although a part of it is split up, still appears in the feces completely undiminished in amount. The fat of milk, 30 to 50 per cent. of which is absorbed, forms an exception. The splitting of the fat also, which takes place without the pancreatic juice, must probably be explained by the action of micro-organisms.

[1] *Zeitschr. f. klin. Med.*, Bd. xii. [2] *Ibid.*, Bd. xvii. [3] *Diss. Strassburg*, 1890.

CHAPTER VII.

EXAMINATION OF THE URINARY APPARATUS.

This comprises the *examination of the urinary organs* themselves and the *examination of the urine*. Indeed, in very many cases the latter examination only is made or it forms the chief part, whether in its relation as being the secretion of the kidneys, or whether it be in reference to admixtures or alterations of the urine which occur in the course of its transit through the urinary passages. The local examination of the urinary organs is now not often required, but if it is, the result of the examination generally confirms the diagnosis. This direct examination, therefore, ought never to be neglected. Moreover, where the kidneys themselves are diseased there come into consideration certain resulting phenomena in the different organs of the body.

EXAMINATION OF THE KIDNEYS.

Anatomy.—The kidneys, about 10 to 12 cm. long, about 5 cm. broad, of well-known form, lie upon the two sides of the spinal column, upon the anterior surface of the quadratus lumborum muscle and the lumbar portion of the diaphragm, and reach from the level of the twelfth dorsal vertebra to the level of the second or third lumbar vertebra. The lower portions diverge somewhat downward, and hence lie with their lower ends somewhat farther from the median line of the body (about three fingers' breadth) than the upper ends (about two fingers' breadth). The right kidney is a little lower than the left.

The upper half of each kidney is covered by the eleventh and twelfth ribs, the extreme upper portion also by the complementary pleural sinus (see Fig. 136); hence the lower border of the lungs does not extend so low as the kidneys. It is very important to note that the outer border of each kidney corresponds tolerably exactly with the outer border of the thick fleshy layer of the sacro-spinalis muscle.

The left kidney at its upper end, rather by its suprarenal capsule, is in contact with the spleen; the right kidney, with the under surface of the liver. Both organs encroach upon the upper end of the kidney of their respective sides, like the tiles of a roof (see Fig. 136). The figure also furnishes information regarding the so-called spleen-kidney and liver-kidney angle.

The anterior surface of each kidney is covered by the parietal peritoneum, and in front of it lies the ascending or descending colon. The anterior inner border of the right kidney is not far from the ductus choledochus and the duodenum.

In the rare condition known as *horseshoe kidney*, the lower ends of the two kidneys are connected by a transverse band consisting of kidney-parenchyma. This transverse portion passes, like a bridge, across

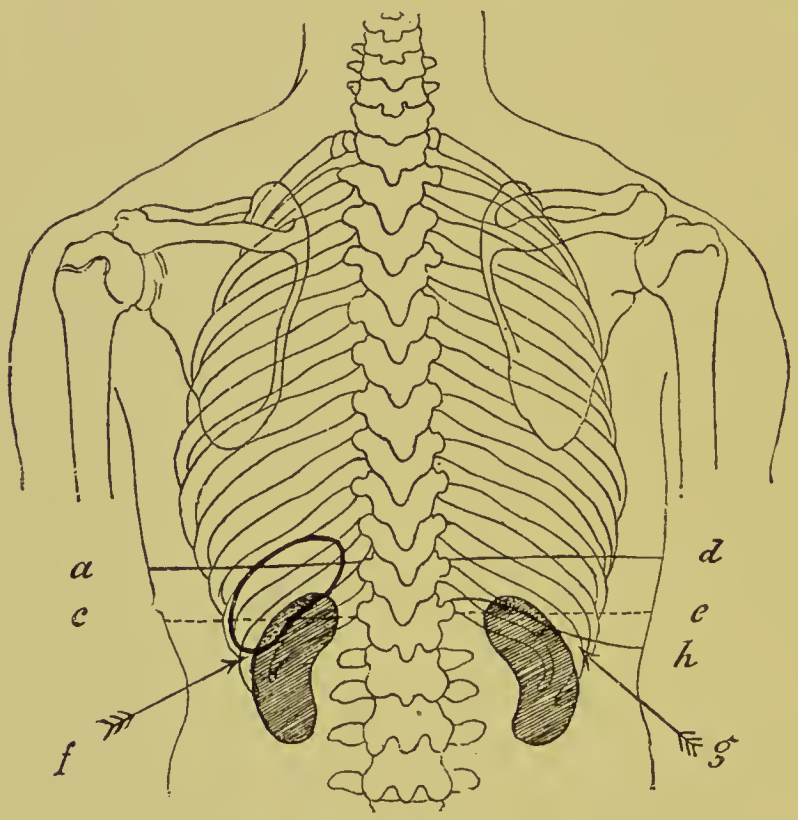


FIG. 136.—Anatomical situation of the kidneys (Weil).
a, *d*, borders of the lungs; *c*, *e*, limits of the pleural sacs; *f*, angle between the spleen and kidney; *g*, angle between the liver and kidney.

the aorta and the spine, about on a level with the second lumbar vertebra.

Local Examination of the Kidneys.—In every respect its result is almost negative.

Inspection.—The normal kidney, of course, cannot be inspected. In remarkably exceptional cases we may, by employing bimanual *palpation*, with the legs drawn well up (one hand being placed behind in the lumbar region and the other pressing deeply in front), get some information, provided the abdominal covering is very unusually lax and thin and the stomach is empty. Of late *percussion* of the kidneys has very rightly come more and more into discredit. It must be perfectly evident that it is impossible to point out the normal kidneys, or even moderately enlarged ones,[1] if one remembers that the kidney is less voluminous than the spleen; that, moreover, it lies much less favorably; and, besides, if he takes into consideration how often the normal spleen is with difficulty or cannot at all be made out. The kidney is unfavorably located for percussion, because the sacro-spinalis muscle (of considerable mass) lies over it, but especially for the reason that its lateral border almost exactly corresponds with

[1] See below.

the convex border of the kidney. So we cannot with certainty determine whether the kidney lies under the muscle nor where its limits are.

Individual exceptional cases, where very thin or atrophic sacrospinalis muscles permit of percussion of the kidneys, may nevertheless occur, as the cases mentioned above, where the normal kidneys can be felt. But we cannot consider the result of percussion of the kidneys as of great value.

We know of a case where a movable tumor in the left side of the abdomen was pronounced to be a floating kidney by a recognized master of percussion on account of a tympanitic resonance in the left renal region. The patient had been carefully examined innumerable times. An operation was performed on account of intolerable pain, and a floating spleen was found. It was removed, however, with a lasting favorable result.

Pathological Conditions of the Kidneys.—Inspection.—The kidney can only be inspected when it is very much enlarged or enlarged and displaced. Tumors of the kidney may make their appearance in the lumbar region, in the side, and in the lateral anterior portion of the abdomen near the border of the ribs. According to their nature, they are smooth, roundish, irregular, or uneven.[1] They do not move with respiration. Their appearance may strikingly vary, but not necessarily so, with the changes of position of the body (the dorsal position, standing). If the tumor is very large it generally presses the colon, ascending or descending, toward the anterior abdominal wall, and then the colon, according to the amount of its distention, may lie up against the abdominal wall.[1]

If the kidney is the seat of a tumor, it very often departs from its place close against the diaphragm and becomes the so-called wandering kidney. In this case it is much easier seen from in front. A normal kidney wandering so much as to be visible is a curiosity (Bartels).

A roundish, symmetrical swelling located in the dorsum in the region of the kidney or somewhat sidewise from it points to *purulent perinephritis.* Sometimes it extends upward in the abdominal cavity, from the diaphragm being pushed up. Often there is edema of the skin at the spot (deep formation of pus[2]) or there may be inflammatory redness. Moreover, abscess, due to the congestion accompanying caries of the spine, may break here. Also, large perinephritic abscesses have been seen as tumors above the border of Poupart's ligament in the iliac region.

Palpation.—This is most important in the local examination of the kidneys. We employ it in the dorsal position with the knees well drawn up, but sometimes also in the abdominal position. In both cases we always first examine bimanually, one hand being upon the region of the kidney and the other upon the abdomen.

Tenderness upon pressure occurs: sometimes in acute, almost never in chronic, *nephritis;* also in *tumor of the kidney, stone* in the pelvis of the kidney if it excites inflammation; in inflammatory *hydronephrosis* and in *perinephritis* (here there is often very great sensibility).

[1] See Palpation.

[2] See p. 48.

When the *kidney* is *enlarged* from engorgement, amyloid disease, or nephritis (large white kidney) it is never perceptible to palpation except it leave its place,[1] or we have one of the exceptional cases in which even a kidney of normal size and location can be felt.[2] Very large new formations, as carcinoma, sarcoma, hydro- and pyonephrosis, echinococcus, and perinephritis only are palpable. The tumor can be felt in one side of the lumbar region or at one side of the anterior abdominal region. With new formations it is unusually uneven; in hydronephrosis, smoothly round, more or less tense; under some circumstances fluctuation can be distinctly made out. Echinococcus is usually smooth and tensely elastic; it may show hydatid vibration.[3]

It is important to remember that tumor of the kidney is only very rarely *movable upon pressure* (for if it descends, then we have a wandering kidney). We have never seen a case where one moved with respiration, but it seems that in some cases there is this movement. At any rate, the absence of respiratory movement points to the kidney, and especially against the spleen or a tumor fixed to the liver.

In a considerable number of cases it will be found that the ascending colon and descending colon are in front of the kidney-tumor and pressed by it against the abdominal wall. In these cases this fact has great value for differential diagnosis. In other cases the tumor will be found lying exactly in the median line, and then it is of significance for differential diagnosis, especially from ovarian tumor. The location of the colon, moreover, is usually only made out with certainty when it can be felt, and particularly when it contains air. It is therefore advisable to inflate it.[4]

Floating Kidney; Movable Kidney.—By this we understand downward dislocation of the kidney, whether much or little. Frequently a dislocation is a partial phenomenon of a general *enteroptosis*. Almost always only one kidney, usually the right one, is dislocated. In these cases the kidney is commonly of normal size, but it may be enlarged, and this is most frequently due to hydronephrosis caused by the bending of the ureter, or also because it is the seat of a new formation.

It is generally very easy to recognize a kidney that is very much out of place, but when it is still high up, near the liver or the spleen, it is often very difficult to do so. The diagnosis is based upon the bean-shaped form of the kidney, eventually upon its being of the appropriate size, and upon its mobility by pressure, which is almost never wanting; also, sometimes, with the changes of position of the body. Not infrequently the kidney can be perfectly replaced. In some cases dyspeptic symptoms, even dilatation of the stomach, also jaundice from engorgement, have been observed when the right kidney was displaced (from compression of the duodenum or of the ductus choledochus. Those cases where the pulse can be felt in the renal artery are rarities.

Percussion.—We employ percussion to establish the existence of *tumors of the kidney*, which give a deadened sound on account of their solidity, but they are almost always clearly made out by palpation.

[1] See Floating Kidney.
[2] See above, Normal Position of Kidney.
[3] See above, p. 292.
[4] See p. 280.

Its value in determining dislocation of the kidney was formerly very much overrated. It was thought that we were able to prove one-sided dislocation of the kidney because, when the patient was lying upon the abdomen, the resonance of the two sides in the neighborhood of the kidneys was found to be different—clearer upon the side of the wandering kidney, in contrast with the absolute dulness of the normal side. In our opinion, even in the most favorable cases, such a condition cannot be employed for deciding the diagnosis.

But, on the other hand, percussion may be of the greatest value, either to determine the relation to the colon of a tumor in one side of the abdomen, or to determine the course of the colon over a tumor of the kidney.[1] In such a case distending the colon with air[2] is of the greatest assistance. Further, it might possibly occur that a considerable enlargement of the kidney could be made probable (never certain) by an area of dulness upon the back extending from the region of the kidneys toward the side.

Differential Diagnosis of Tumor of the Kidney.—The positive evidence of tumor of the kidney has just been spoken of. We may have to make a differential diagnosis between a *wandering right kidney* which is not very much displaced downward and a distended gall-bladder, or an echinococcus located upon the lower surface of the liver. If there is respiratory mobility, this speaks against it being the kidney, but if the tumor can be replaced, so that it may even disappear, then it speaks for it being the kidney. Both wandering kidney and a pedunculated echinococcus may be easily movable upon pressure. It may often be impossible to determine exactly the form of a tumor situated close under the liver.

A *wandering left kidney* is distinguished from a *wandering spleen* by its form, which is made out by percussing the neighborhood of the region of the spleen: in wandering spleen we may find notches; if it is the kidney, we may feel the pulse at the hilus. We distinguish tumor of the left kidney from tumor of the spleen by the form and relation to the colon. Sometimes respiratory mobility decides in favor of the spleen, but this may also be wanting; while notches on the upper border of the tumor may speak with probability for the spleen; yet in one case, where they could be very distinctly felt, they led us to a false diagnosis; it was found to be a carcinoma of the kidney.

EXAMINATION OF THE URETERS AND BLADDER.

Ureters.—Simon, by introducing the hand into the rectum, has repeatedly felt the ureters.[3] Recently, Heger-Kaltenbach and Sänger have proposed, in the case of women, to palpate them *per vaginam*. We can feel their lower ends where they come down on either side of the neck of the uterus and enter the lower side of the bladder. With some practice often even a normal ureter, but still more one that is thickened, can be felt in the lateral and anterior *fornix vaginæ* and the anterior vaginal wall close to the middle line.

In this way it is not difficult to recognize *thickening or tenderness* of one or both ureters occurring in cystopyelitis and in tuberculosis of

[1] See above. [2] See p. 280. [3] See works upon Surgery.

the urinary apparatus; thickening and distention may sometimes be observed also in *pyelitis calculosa* (renal calculus).

Bladder.—The bladder lies behind the symphysis pubis; when ordinarily distended it rises above it; but only when it is excessively full, as in paralysis of the bladder, spasm of the sphincter, stone in the bladder, stricture of the urethra, does it swell so much as to be noticed (rarely) by inspection, but especially by palpation and percussion, as a roundish tumor, which of course is dull in sound. In men it can also be felt from the rectum. We are able to decide with certainty whether a tumor in the hypogastrium is a distended bladder or not by drawing off the urine with a catheter. It may be confounded with a pregnant uterus and also with other swellings. Always before undertaking an examination of the abdomen we must see that the bladder is empty, partly to avoid confounding the distended bladder with something else, and partly because, if the bladder is full, it interferes with the examination of the abdomen.

F. Müller has ascertained that it is necessary to have 500 to 600 c.cm. of fluid in the female bladder, and 360 to 500 c.cm. in that of the male, in order to have dulness upon percussion over the bladder.

Anomalies located in the wall of the bladder can usually be felt best when the bladder is full. The external examination is made *per vaginam*, *per rectum*, and sometimes bimanually.

Surgery and gynecology teach the complicated methods of examining the bladder and ureters. With reference to the examination of the male urethra we refer to works upon Surgery.

EXAMINATION OF THE URINE.

Under normal conditions and free from admixture, the urine exhibits the renal secretion in a state of purity only as it issues from the orifice of the ureter, since in its transit through the urinary passages it receives a few epithelial cells and micro-organisms[1] from the mucous membrane—additions, however, that are scarcely worth mentioning. At the time of its discharge from the body and for some time after its physical and chemical conditions are the same as at the moment of secretion. In a number of pathological conditions also the urine is the pure and unaltered secretion of the kidneys; while in a second series of diseases it is changed by its exit from the body by admixtures from the urinary passages, or by decomposition of its constituents in the bladder. To the first series belong the anomalies of the secretion itself; to the second, the diseases of the urinary passages.

In women the urine may be contaminated by admixture of material from the vagina or uterus, and of these the most frequent and important is the menstrual fluid. In order to avoid this contamination we are sometimes obliged to draw off the urine with the catheter. It is usually contaminated by fecal material only from carelessness of the patient or of the attendant. But sometimes it results from communication of the intestine with the urinary passages, as of the rectum with the bladder or with the vagina.

[1] See below.

In examining urine for bacillus tuberculosis it is sometimes necessary to guard against its being contaminated by the patient's sputum.

Recent investigations by Lustgarten and Mannaberg show that the former assumption that the urine is normally free from bacteria must be given up. The urine of healthy persons contains a number of microorganisms which have their origin in the urethra. The most important are a large streptococcus; a diplococcus which resembles the gonococcus, also like that in epithelium, but of course it is not found in pus-corpuscles; and, lastly, a bacillus which morphologically and in its color-reactions agrees with the tubercle bacillus, and which probably is the smegma bacillus, which also occurs in the preputial sac. This latter may give occasion for the erroneous supposition that there is tuberculosis. But that it has its origin in the urethra is shown by the fact that it is observed even when the preputial sac has been most carefully cleaned previous to urination, though it is only found in individual cases, while in cases of tuberculosis it is always abundantly found in the urine. Sometimes inoculation must decide.[1] We may avoid the urethral bacillus by drawing the urine with a catheter, but then also, sometimes, possible tubercle bacilli from the prostate or genital apparatus may be found in the urine.

In case of disease of one kidney or pelvis of the kidney the question may arise as to what part of the urine passed is from the right, and what from the left, kidney. If one kidney fails, the other acts vicariously. In tuberculosis of the urinary passage and in pyelitis it may happen that for a time one ureter is stopped; the urine comes only from the other kidney, and it may be quite normal. Then, suddenly, the character of the urine will change, showing considerable white blood-corpuscles, seed-like particles, tubercle bacilli, or calculi and blood. The quantity of urine is, for the time being, increased, for the closed side has again opened.[2]

In certain diseases of the urinary apparatus *the manner of passing the urine* shows characteristic peculiarities; but in many of the conditions under consideration the urine is passed in a perfectly normal way. Painful strangury, frequent urination, a feeling of burning in the urethra while passing the urine, may result from the urine being much concentrated, such as is passed when there is engorgement of the kidneys and in the majority of cases of acute nephritis. Very pronounced *tenesmus* of the bladder—that is, painful urgency, extremely frequent, very painful urination, in which only a small quantity of urine is passed at a time—indicates *cystitis*. We must mention here, further, retention and incontinence of urine and nocturnal enuresis.[3]

In regard to *the mode of procedure in examining the urine*, let it be here remarked, in the first place, that we should take care that the urine is received into vessels that are perfectly clean—if possible, in glass vessels; and also that for judging of certain general characteristics it is necessary to examine the mixed urine passed during twenty-four hours, or that passed during the day and during the night, separately. For certain examinations it is necessary to separate, in the most careful way, the urine passed each twenty-four hours. In the warm season of

[1] See Appendix.

[2] See page 367.

[3] Regarding these, see under Examination of the Nervous System.

the year the urine ought to be examined as soon as possible after it is passed. [Sometimes the nurse may be directed to add a few drops of chloroform or of a 50 per cent. solution of chloral hydrate to the urine immediately after it is voided. This will prevent decomposition and development of bacteria till it can be analyzed.] In order to examine the sediment the upper portion of the urine is to be carefully poured off, and the remaining cloudy portion is put into a conical glass, in which it is allowed to stand till the sediment is deposited; then with a pipette we take up a few drops from the bottom of the glass.[1]

When there is unconsciousness or difficulty in passing the urine we must employ the catheter. The artificial emptying of the bladder for the purposes of examination must never be omitted in any case of unconsciousness.

The examination of the urine comprises the following points: the *amount* [in twenty-four hours], its *color* and *transparency*, *specific gravity*, *reaction*, *odor*. In addition, the urinary sediments and constituents in solution must be studied. We briefly describe the characteristics of normal urine.

(A) Normal Urine.

1. Amount.—In twenty-four hours, with healthy persons, it amounts on the average to about 1500 grams. But its variations within physiological limits are very considerable, since every increase in the amount of water taken increases the amount of the urine, and every increase in the amount of water disposed of in other ways [by perspiration, respiration, vomiting, and diarrhea] diminishes the urine. In the latter respect, in health we have to consider the loss of water by respiration and by perspiration from heat and from active bodily exertion. It is superfluous in the cases just referred to to specify the maximal and minimal figures for the amount of the urine; only when those conditions are wanting must a departure from the average quantity of urine given above cause us to think of a pathological condition.

Within the twenty-four hours the least urine is passed at night or in the early morning, very much the greater portion being passed during the course of the day. Quincke estimates that the amount of urine excreted hourly in health during the night, compared with the amount during the day, is in the proportion of one to two or three. Usually, the amount of urine passed increases about an hour after taking fluid. Emotional excitement, especially anxiety, sometimes temporarily increases the secretion of urine.

2. Color; Transparency.—In health the color is usually dark straw-color to reddish-yellow. Generally, the greater the amount of urine the clearer it is. In this respect, as well as in the quantity, with physiologically exceptional cases, it shows marked variations from the average—from being almost as clear as water after a great amount of fluid has been taken, to a decidedly dark reddish-yellow (concentrated urine) after severe sweating. The coloring materials which give the normal color to the urine are not yet all exactly known. The most important pigment seems to be *urobilin*; moreover, *indican* interests

[1] Regarding the use of the centrifuge for shortening this part of the process, see page 374.

the clinician. Both coloring materials may, in disease, be pathologically increased.[1]

Urine freshly passed, in health, is always perfectly clear and transparent, but in these respects it may change some time after it has been passed.

(*a*) In almost all normal urine, after standing a short time, there is formed a slight cloud of mucus (*nubecula*). It consists of a mucin-like substance which in part is probably mucin, in part an albuminous substance related to it, nucleo-albumin. Both substances are present in every specimen of urine, part of which is precipitated as a cloud of mucus, but part also remains in solution. This is from the urinary passages, chiefly from the bladder. It is increased, not only in many diseases of the urinary apparatus, particularly of the urinary passages, but also in health. As it is precipitated by some of the albumin tests, it may simulate genuine albuminuria.

(*b*) It not infrequently happens, with healthy persons, that the urine, if somewhat concentrated, is cloudy when it becomes cool, from the separation of the uric-acid salts. Gradually the salts sink and form a sediment of clear brickdust-red or flesh color (associated coloring matter of the urine, *brickdust sediment*, *lateritious sediment*). It has the peculiarity—by which it is likewise recognized—that it is again immediately dissolved as soon as the urine is warmed. After a long march in the heat this sediment occurs very regularly, because the urine is then concentrated, but it also is observed in urine that is not so very dark if allowed to stand in a cool place.[2]

(*c*) Urine that stands exposed for a long time, both clear and dark, likewise sometimes becomes cloudy because it undergoes *ammoniacal fermentation*. The urea is changed into carbonate of ammonia, which makes the urine alkaline, whence there is a deposit of *phosphates* (ammonio-magnesian phosphates or triple phosphates, also phosphate of lime). Urate of ammonia also is formed and deposited. These separations and numerous bacteria render the urine cloudy and gradually form a *whitish sediment*. In hot weather this ammoniacal fermentation takes place within a few hours after the urine is passed; in a cool place it does not begin before thirty-six to forty-eight hours, or not at all.[3]

3. Specific Gravity.—In health it usually varies between 1015 and 1020. It depends upon the amount of solids held in solution by the urine; hence, on the one hand, upon the absolute quantity of the solids, and, on the other, of the amount of the watery portion of the urine or the quantity of the urine. The abundant urine which follows drinking a great amount of water is always of low specific gravity, and therefore clear. A scanty urine, from the loss of water in other ways, is always of high specific gravity, and hence is dark. Then, also, in health the specific gravity, under some circumstances, temporarily oversteps very considerably the figures given above—from as low as 1003 to as high as 1025, or even higher. In the absence of "physiological causes" these figures are always of pathological significance.

[1] See Pathological Color of Urine. [2] See further regarding the Urinary Sediments, p. 386.

[3] For a more particular account of the condition when there is ammoniacal fermentation of the urine, see p. 373.

Mode of determining: We measure the specific gravity of the urine by means of an aräometer, graduated for taking the specific gravity of the urine (that is, from 1000 to about 1040—urinometer). We take a portion of the urine which we wish to weigh (generally a mixture of that which has been passed during the previous twenty-four hours) and pour it into a not too narrow cylindrical glass until the column of urine is longer than the urinometer. With filter-paper or a pipette we remove any air-bubbles from the surface, and then introduce into it a perfectly clean and dry urinometer, wait until it has become quiet, and then observe the figure that stands opposite the lower border of the meniscus of the fluid. [It should not be read from below the surface of the fluid, but from above. The division of the stem nearest the general level of the liquid is the one to select.]

[To overcome the inaccuracies which inhere in most urinometers Dr. Squibb has proposed two important modifications, that the air-chamber of the hydrometer should be that of a double cone, and that the jar containing the urine should have its sides indented so as to make an inverted V-shaped projection inside the jar. Both of these reduce to a minimum the friction between the surfaces.[1]]

None of the simple medical instruments is so often useless as the urinometer. We should never use one until its accuracy has been tested. It is always desirable to have a urinometer upon which is given the temperature for which its scale is arranged; not that we must always have the urine at this temperature, but because the absence of this declaration from the instrument shows very certainly that it has been prepared without care. The scale is arranged for a temperature of 15° C.—*i. e.* about room-temperature. When the temperature of the urine differs considerably from this—for instance, when it has been recently passed or has been kept in a cold room in winter—considerable error results. Then it is necessary to add or subtract one degree on the urinometer for each three degrees Celsius which the temperature of the urine is less or exceeds that prescribed for the urinometer.

4. Reaction.—In general this is *always acid*, chiefly from the presence of acid urates and phosphates. The degree of acidity varies individually; moreover, it is a constant quantity in every individual case of health and when the food is approximately alike.

But in the twenty-four hours the reaction varies considerably, so as to be even alkaline and yet physiological. The variations proceed in such a way that after every meal consisting of a mixed diet the acidity declines until, after about two hours, it becomes alkalescent; but this quickly passes, so as to give place again to an acid reaction (Görges). These variations have been referred by many to the loss by the body of acids and alkalies in stomach and intestinal digestion. Hence it is assumed that the separation of HCl in the stomach increases the alkalescence of the blood, and hence the urine becomes less acid, or alkaline. But, according to recent investigations by v. Noorden, this increased alkalinity of the blood does not exist. By a graphic representation of the reaction of the urine during twenty-four hours we obtain the so-called "acid-curve." This, with some healthy persons and under like

[1] See Squibb's *Ephemeris of Materia Medica*, vol. i. p. 357 *ff*.

conditions as to time and quality of food, is tolerably constant, but with other healthy persons it varies considerably.

Sometimes the reaction of the urine is *amphoteric*—that is, it colors red litmus blue, and at the same time colors blue litmus red.

The neutral or alkaline urine of health at the time of passing is usually clear. But it quickly becomes cloudy from the withdrawal of the phosphates, which gradually form a sediment. The cloudiness does not disappear upon the application of heat, but becomes more marked; on the other hand, the urine again becomes clear upon adding acetic acid, in consequence of the solution of the phosphates.

5. Odor.—The normal aromatic odor of urine is well known; it is changed by certain foods. Most frequent and most striking is the stench of urine after eating asparagus; garlic gives its odor to the urine. During alkaline fermentation we may have the development of ammonia, which gives its known pungent odor.

6. Sediments.—With reference to the cloudiness the urate sediment of the acid and the phosphatic sediment of the alkaline urine have been mentioned on page 363. Regarding the microscopical condition of the sediment, see page 360. By the use of the centrifuge it has been found that isolated casts and red and white blood-corpuscles are present in the urine of persons who show no other morbid signs, particularly also no chemically demonstrable albuminuria.

Whenever there is a sediment it is not unimportant to remember that different things may have been mixed with the urine after it was passed.[1]

7. Micro-organisms.—Regarding these, see what has been said above, page 361.

8. Urinary Constituents in Solution.—The constituents of normal urine besides the coloring-materials, which, from our present knowledge, are of importance to the clinician, are the following: urea, uric acid, creatinin, oxalic acid, chlorid of sodium, sulphates, phosphates, carbonates.

Urea $\left\{ CO\begin{matrix}NH_2\\NH_2\end{matrix} \right\}$ passed in twenty-four hours amounts in the adult to about 30 grams (men somewhat more, women somewhat less). However, the amount of urea varies within wide limits: it is dependent upon the amount of albuminous material in the food taken, and, on the other hand, it is usually independent of the amount of muscular exertion.

Uric acid, like urea, is a product of the metabolism of albumin; in man the quantity is much smaller than the former, being in proportion to the urea about as 1 : 45; but it is to be remarked that great variations take place, chiefly under the influence of the food, and this in such a way that albuminous food increases the acidity of the urine. With reference to clinical diagnosis, the uric acid and also the creatinin are chiefly of interest, because they may place difficulties in the way in examining the urine for sugar, in that they sometimes simulate the reaction of sugar. Sometimes, on the other hand, they hinder the reaction of sugar.[2]

Chlorid of sodium, the most important of the inorganic constituents,

[1] See above, page 360.

[2] See under Mellituria.

in health corresponds in amount with tolerable exactness to the amount of salt in the food taken. On the average, it usually is proportioned to the urea as 1 : 2 to 1 : 3.

Exceptionally, in health, there are found in the urine the following:

Albumin, the so-called physiological albumin. There is still great difference of opinion regarding this subject: while it is doubted by some, others maintain (Senator, recently Posner) that traces of albumin exist in the urine in every healthy person. It occurs in very small quantity (about 1 per cent.) after severe exertion or hearty eating. The urine of the newly-born not infrequently contains some albumin (compare what has already been said on page 363).

Sugar may occasionally appear in small quantities in the urine (0.1 to 0.25 per cent., Moritz) after over-abundant use of food and drinks containing sugar (confectionery, etc., sweet champagne, even great quantities of beer). This alimentary glycosuria is possibly favored by individual disposition. At the same time, however, it seems that we are not justified in considering persons who show these signs after indulgence in sweets as disposed to diabetes. Sugar of milk to the extent of 0.8 to 1, even 2 per cent. appears during confinement and also lactation.

Reducing substances, which are not sugar, are present in every normal urine, but only exceptionally can they be demonstrated by the common tests. The reduction is very slight, perhaps about 0.1 to 0.22 per cent. solution of grape-sugar (Moritz). Generally, the quantity of reducing substances seems to go parallel with the excretion of nitrogen. Uric acid and creatinin take the principal part in the reduction.

Bile-acids are likewise observed in very small quantities in normal urine.

Fat is recognizable generally only in microscopical drops (or only in ether extract), and is found when the food has contained a great abundance of fat, as of cod-liver oil.

(B) Pathological Urine.

Anomalies in the Quantity.—Increased amount of urine (*polyuria*) is observed—

1. In a *watery condition of the blood* in the different forms of anemia or hydremia. The increase here is never very great: 2000 grams or less; there may be no increase, and if the heart is weak it may even be diminished.[1]

2. In the different forms of *contracted kidney*, and this in consequence of the accompanying hypertrophy of the left ventricle, which causes increased pressure in the whole arterial system, and thus also in the renal arteries: here even to 3500 grams or more. Here the chief cause of the polyuria is the increased arterial pressure from the increased action of the heart.[1]

3. When the *exudation or transudation* in the serous cavities of the body or the fluid in the cellular tissues (edema) is *resorbed*, the daily excretion of urine sometimes amounts to 4000 grams or more. The increased arterial pressure[2] from quickening of the action of the heart,

[1] See page 368.

[2] See page 368, under 3.

which occurs at the same time, is also a prominent factor in producing polyuria.

4. In *diabetes*. Both diabetes insipidus and mellitus (mellituria) manifest themselves by the increase, often an enormous amount of urine, 4000 to 10,000 grams and more. Sometimes in diabetes mellitus there is only a moderate polyuria, or, for a time, in this disease there is even complete absence of polyuria (diabetes decipiens).[1]

5. *Nervous polyuria* (urina spastica, nervosa). This may occur temporarily or paroxysmally in attacks of migraine, epilepsy, in psychical excitement of nervous persons, but it may also be of longer duration, as in hysterical neurasthenic people. Sometimes there is a periodical flood of urine having a nervous origin.

6. As a necessary consequence of abnormal thirst, *polydipsia*, as it is sometimes observed, particularly in hysteria.

7. A *periodic* (or better, a temporary) *flood of urine*, which occurs only once in a while, is sometimes observed in conditions which suggest a temporary exclusion of one kidney.[2] These are cases of tuberculous and catarrhal ureteritis with obstruction of the ureter by swelling of the mucosa, detritus, etc., also nephrolithiasis (which, however, is always accompanied by colic, and, lastly, floating kidney, if its movements cause a bending of the ureter. During the occlusion of the ureter there is hydronephrosis, and the flood of urine makes its appearance as soon as the obstacle disappears.[3]

Finally, we must briefly refer to some *drinks* which temporarily increase the amount of the urine, as coffee, beer, and wine, which increase the quantity of urine more than the amount of water represented. Likewise, there are to be mentioned certain medicines which have the same effect, partly in that they increase the blood-pressure by affecting the action of the heart, partly in that they stimulate the secreting action of the kidneys.

In the above pathological conditions, where we do not have a removal from the organism of water that has accumulated there, the polyuria must, of course, be made up by imbibing an increased amount of drink (*polydipsia*). Whether we have the increased thirst from increased loss of water, or whether the polyuria is the result of the polydipsia, is not entirely clear, especially in many cases of diabetes insipidus. In diabetes mellitus the polyuria is probably only a purely secondary result of the polydipsia, which, in turn, is to be regarded as the consequence of the glukemia (Cohnheim).

Diminished amount of urine, under some circumstances even to the extent of not passing any (*anuria*), occurs—

From diminution in the secretion of urine—

1. In the *loss of water in other ways:* in severe sweating[4] in any kind of severe diarrhea, particularly in *Asiatic cholera*, where for days together there is continuous anuria. Thus, also, during the formation of a *pleuritic or peritoneal exudate*, where fever is also to be taken into account as a cause.

2. *In fever*, and largely in consequence of the loss of water in other

[1] See under Specific Gravity and Sugar in the Urine.
[2] See page 361.
[3] See page 358.
[4] See also Normal Urine.

ways, by increased perspiration and the greater loss of water by the lungs.

3. By *reduced blood-pressure* resulting from the diminished work of the heart: hence in diseases of the heart-muscle; incompensation in valvular disease; in weakening of the hypertrophic heart of contracted kidney; in emphysema; in all the diseases, frequently mentioned, which harmfully affect the action of the heart. In these conditions the amount of the urine is the chief means of forming a judgment of the course of the disease and furnishes the indications for treatment.

4. In *acute*, *subacute*, and *chronic nephritis*, except contracted kidney (regarding which, see also above under 3). In these diseases also the amount of the urine is a symptom which indicates the severity of the case. In acute nephritis, not infrequently, for a time there is anuria.

5. From suppression of urinary secretion due to nervous causes, especially in a still indistinct reflex way in trauma, as from operations, affecting the abdomen.

Also, there may be a less quantity of urine from *difficulty in micturition:* from a very narrow stricture of the urethra (surgery); from retention in the bladder; from obstruction in the ureters. In regard to the latter, when one kidney is cut off the other generally vicariously performs the work of both; but there may also be anuria when one ureter is closed, as from stone in the kidney, and this, in fact, from a kind of reflex suppression in the other kidney ("shock").

The great zeal in using the catheter in recent times has given us as a result, among other things, the knowledge of the fact that in health with every urination the bladder is completely emptied, even to a few drops. If a certain amount of urine remains in the bladder (*residual urine*), there is a pathological cause for it. This may be a purely mechanical hindrance to the emptying of the bladder, as stricture, hypertrophy of the prostate, urinary calculi; or it may result from the mechanical hindrance—atony of the bladder; or there may be primary nervous paresis of the detrusor, as occurs in *tabes* and in *all diseases of the lumbar cord*. The amount of residual urine is said to be tolerably constant; it is measured by having the patient pass his urine and then use the catheter immediately afterward.

Color and Transparency of the Urine in Disease.—Color.—Primarily, the color varies according to the degree of concentration, in the same way as in normal urine; and as in health, so also in general in disease, it stands in a certain relation to the amount of the urine: the greater the amount the clearer the urine. But, like the variations of quantity from the average, the changes in the color of the urine (apart also from unusual colors)[1] are also much more significant in disease than is the case in normal urine. The scale of colors of the urine passes from the almost colorless to the straw-yellow, reddish, red-brown, even brown-black. It is not necessary to have a very exact determination of the color of the urine by comparing it with those of a table of colors, as was proposed by Vogel, because it could only have a value in determining the degree of concentration, and generally for this the specific gravity is much more exact.[2]

[1] See next page.

[2] See Specific Gravity.

Patients with cirrhosis (N. B., without icterus)[1] sometimes pass urine that, in proportion to its amount, is very dark. Anemic (chlorotic) persons, on the other hand, often pass remarkably clear urine. In fever the urine is relatively dark—reddish or brownish-red (urobilin).[1]

In *diabetes mellitus* there is a peculiarity in the very striking contradiction between the clear color and great amount of the urine on the one side, and its high specific gravity upon the other, which is of diagnostic importance.

As *special pigments* of the urine, the following are to be mentioned:

1. Color due to the increase in the normal pigments. Two of these come into consideration here:

Indican, occurring in increased amount, may sometimes give to the urine a bluish or bluish-black color, if it has been decomposed in the urinary passages and changed into indigo-blue; but very often we do not recognize that the urine contains more indican, because indigo has not yet been formed. Hence, when there is a suspicion of indican, or if we wish to make use of its possible presence for the purposes of diagnosis, even when the urine appears to be perfectly normal, we must examine it with reference to this substance. When urine containing indican has been standing for some hours, it can generally be recognized by the bluish shimmer of the residuum from the drops of urine from the upper part of the urine-glass sprinkled and spread out as thin as possible, and sometimes, also, by a bluish film upon the surface of the urine. Besides, all of the urine is sometimes blackish-blue, and this is most markedly the case when the urine putrefies.[2]

Indicanuria—that is, increase of the indican—occurs: when there is accumulation of the intestinal contents, especially of the contents of the small intestine, hence in occlusion of the intestine from any cause, as peritonitis or obstinate obstipation; likewise, in all forms of severe cachexia, as well as in Asiatic cholera; lastly, in individual cases in health. Children are generally inclined to an abundant excretion of indican.

Urobilin, if it exist in considerable quantity in the urine, colors it a decided red or brownish-red. The foam of the urine sometimes looks yellowish-red or yellowish-brown. Moderate quantities only indistinctly discolor the urine. While there is only a small quantity of it in health, it is abundant in febrile diseases, especially in alcoholics, and where there is at any time resorption of large effusions of blood, and in the most varied affections of the liver (as a first sign of alcoholic cirrhosis, in the first stage of congestion of the liver—Hayem). When there is a marked separation of it which continues for some time, a brownish discoloration of the skin is observed in the so-called *urobilin-icterus*, though there is still dispute as to its nature (compare what has already been said on page 42).

Proof of the increase of indican: The following reaction (v. Jaffé) establishes the presence of indican in increased amount, because it does not operate in the presence of the small quantity found in normal urine. We mix equal parts of urine and fuming hydrochloric acid in a reagent glass; into this we drop two to three, or at most four, drops of

[1] See below.

[2] For its Chemical Reaction, see next page.

a concentrated solution of chlorinated lime: immediately, or after a few seconds, there is formed just beneath the surface a blue-black cloud—indigo-blue. By stirring the solution of lime in the urine we obtain, according to the quantity of indigo formed, a more or less dark coloration of the whole fluid. If, then, we add a few drops of chloroform and agitate (not shake) the reagent-glass several times, we have the blue color at the bottom from the settling of the chloroform. Though there may be an increase of indican, it will not be shown by the reaction if too much chlorinated lime has been added, because the indican is then oxidized into indigo-white.

Obermayer has lately suggested an indican test which seems to be more distinct and reliable than Jaffé's. It is also somewhat more circumstantial. To the urine 20 per cent. of sugar of lead is added; then filter; to the filtrate add an equal volume of fuming hydrochloric acid containing 0.4 per cent. of chlorid of iron. Shake the mixture, when the reaction will develop in two to three minutes. After this apply the chloroform as above.

Proof of urobilin. 1. Spectroscopic: Absorption bands in green-blue, between Frauenhofer's lines b and F (sometimes it is necessary to dilute the urine with water, in order to be able to make the examination). Tissier has lately recommended to superimpose the urine with water. The urobilin rapidly diffuses into the water, and in this it is easily recognized by the spectroscope. 2. Chemical: We add ammonia to the reddish urine in the reagent-glass. If there is much urobilin, it gradually becomes a clear green; it is then filtered; and, sometimes, upon the addition of a few drops of a watery solution of chlorid of zinc, there appears the rose-red-greenish fluorescence that is peculiar to urobilin.

2. Discoloration of the urine from the presence of the coloring-matter of the blood, and of the bile. Coloring matter of the blood colors the urine variously according to the amount that is mixed with the urine, also whether it is fresh or has been changed, and according to the original color (concentration) of the urine: flesh-red or blood-red with greenish shimmer with the light passing through it, corresponding to the dichrotic behavior of the blood; or an untransparent brown, even blackish-brown. Frequently the bloody color is easily recognized; but, generally, the reaction-test for blood coloring matter is necessary.[1]

Coloring matter of blood occurs *in the urine:* 1. In *hematuria.* Its occurrence here is circumstantially described in the section on admixture of blood with the urine in connection with Urinary Sediments, on page 375. 2. In *hemoglobinuria.* In this condition the hemoglobin is found entirely dissolved or in granular lumps, but no red blood-corpuscles, or very few, are found in the urine. This results from hemoglobinemia (see pages 233 and 235), and this condition may arise from very different causes: from poisons (chlorate of potash, mineral acids, arsenical solutions, pyrogallic acid, naphthol, poison of the edible mushroom, *helvella esculenta;* after transfusion of animal blood (as of lamb's blood); in infectious diseases (as scarlet fever, abdominal typhus [typhoid fever], malaria, syphilis); after extensive burns; lastly,

[1] See p. 399.

we have to mention a form of hemoglobinuria which occurs as an independent disease—*paroxysmal hemoglobinuria.*

Coloring matter of the bile exists *in the urine* in icterus (icteric urine). Such urine is most frequently a beer-brown, sometimes brown-green, or even black. If the urine of icterus, as is very seldom the case, is very thin, then it may have a golden-reddish tone. The foam that forms when it is shaken is then highly characteristic: from clear to dark yellow, green-yellow, even brownish. Regarding the chemical tests for bile coloring matter, and more particularly regarding its presence and that of the bile acids in the urine, see section on Coloring Matter of the Bile, page 363.

3. Discoloration of the urine from medicines. It is very important to recognize these changes in color, so that one may be on guard against deception by confounding them with the coloring matter of the bile and the blood.

The chrysophanic acid contained in *rhubarb* and *senna* passes off by the urine. It colors the urine slightly, making it at most a little brownish, if normally acid; but if alkaline, or is made so, then it becomes a purplish-red.

After taking *logwood*, alkaline urine also becomes reddish or violet.

Santonin colors the urine yellow or greenish-yellow, with a yellow foam; upon the addition of an alkali the color changes to red. *Picric acid* makes the urine yellow, but there is no change in color after changing the reaction.

Carbolic acid, naphthalin, creasote, and other coal-tar preparations, as well as the infusion of the leaves of uvæ ursi (arbutin), produce a greenish or greenish-black color of urine.

Brownish or blackish discoloration of the urine after standing for some time in the air is observed in patients with melanotic tumors, because the pigment which forms the coloring matter of the blood in those tumors passes off by the urine. A similar behavior of the urine is found in the presence of an abnormal amount of pyrocatechin, an extremely rare occurrence.

Transparency of the Urine.—A loss of transparency by turbidness may take place even in normal urine when it has been allowed to stand.[1] Likewise a more marked cloudiness (nubecula) occurs in normal urine from the increase of the mucin-like substance. This also occurs in diseases of the urinary apparatus, particularly of the urinary passages. Urine that is turbid when passed is always pathological. This is the case: first of all, in *nephritis*, in consequence of the presence of organized constituents; in all diseases of the urinary passages, for the same reason (here particularly on account of mucus); but especially in severe *cystitis*, because the urine in this condition is alkaline when it is passed (alkaline fermentation in the bladder), and hence, besides the organic constituents, contains a deposit of phosphates. Admixture of *blood* and *pus* always makes the urine turbid to some extent. The most striking and, at the same time, the rarest kind of turbidness is that caused by fat in the urine—*chyluria.* Here the urine is milky, as if mixed with pus (*galacturia*) from the emulsified fat; or it contains large drops of fat or fat-bubbles swimming upon its surface

[1] See p. 363.

(*lipuria*). By shaking the urine up with ether it becomes clear. But when it is allowed to stand, part of the fat settles as a sediment, and part forms a cream-like layer on top.[1]

The Specific Gravity of the Urine in Disease.—The specific gravity of the urine may vary from a little over 1000 to over 1060 (in diabetes mellitus). Apart from certain special admixtures (we mean particularly sugar, which increases the specific gravity without changing the color, and the special pigmentary admixtures, which, on the other hand, darken the color without essentially adding to the specific gravity), almost always in disease, as in health, a scanty, dark urine has a high specific gravity; an abundant, clear urine, a low specific gravity. According to Haeser and Neubauer, from the specific gravity we can obtain an approximation to the amount of solid constituents of the urine by multiplying the last two figures of the specific gravity by 2.33. This product represents the quantity of solid constituents in 1000 grams of urine. If we have 1200 grams of urine with a specific gravity of 1021, then 1000 grams of this contains $21 \times 2.33 = 48.93$ grams of solids, and the whole amount = 58.7 grams. But not much has been said regarding the change of material upon which it chiefly depends, because the different solid constituents of the urine have very different specific gravity, particularly urea, which, as compared with sodium chlorid, is as 2 : 3. Hence, we can never draw definite conclusions from the specific gravity alone, and even where we can exactly determine the solids, as by examining the various material changes, *the quantitative determination of the urea* or of the nitrogen is indispensably necessary.

The chief value in the determination of the specific gravity with reference to diagnosis consists in the following:

1. High specific gravity with clear and abundant urine points to *diabetes mellitus*. We may even say that a specific gravity of 1040 and over, the urine being clear, can only be caused by sugar, and hence is pathognomonic of diabetes.

2. Repeated or continued examination of the urine in general engorgement is of value, because this, as well as the quantity of the urine, measures the labor of the heart.

It is not unimportant to know further—

3. A low specific gravity, when there is a small amount of urine which is often high colored, occurs in *nephritis* from diminished excretions of urea, also in severe diarrhea and vomiting.

Reaction of Urine in Disease.—For the reasons previously given[2] the reaction of the urine is reliable only a short time after it has been passed.

Neutral or alkaline reaction of the urine is met with in sickness—

1. Under the same conditions that make it neutral or alkaline in health.

2. When there is resorption of transudates and exudates in the cavities of the body, also from large effusions of blood, especially in the pleura and peritoneum.

3. With dilatation of the stomach, and particularly if the contents of the stomach must frequently be brought up, either by vomiting or arti-

[1] Further regarding Chyluria, see p. 378.

[2] See p. 364.

ficially. The reason given is that the blood and the organisms lose their acidity because free HCl is not again resorbed (?).[1]

4. Considerable admixture of blood or pus. In the cases of alkaline urine previously mentioned the urine is clear, or is turbid from the deposit of phosphate; it contains no bacteria or only a few.

5. With alkaline fermentation of the urine in the bladder. This accompanies severe forms of *cystitis*. Here the urine is turbid because of the presence of pus-corpuscles, abundant bacteria, deposit of triple phosphates, urate of ammonia, carbonate and phosphate of lime and magnesia. Sometimes it has a peculiar urinous smell, and is pungent from the free ammonia. By this latter a strip of red litmus-paper, just held free over the fluid, is colored blue.

Further, regarding the formed constituents of simple alkaline urine and that which has been the subject of alkaline fermentation, see under Sediment.

The *acidity of the urine* may be *determined* by a simple but really not very accurate method: Prepare a 10 per cent. solution of caustic soda (1 of soda to 9 of distilled water), and pour this from a burette into the urine until a piece of very sensitive litmus becomes blue: 1 c.cm. of the soda solution corresponds to 0.0063 of oxalic acid.

Works upon analysis of the urine teach the more exact methods.

Pathological Odor of the Urine.—Here we must mention as worthy of recognition the pathological departures from the odor of normal urine. A urinous, more or less pungent, ammoniacal odor in cases of severe cystitis shows ammoniacal fermentation in the urine that is passed. Then there is the feculent odor when the urine is mixed with feces, whether the admixture takes place after the urine is passed,[2] or whether it has taken place from communication between the bladder and the intestine, with discharge into the bladder.

The most notable, and at the same time diagnostically important, odor of the urine is the fruity (*apple-odor*), or like chloroform. The substance which has this peculiar odor seems to be acetone (Petters) [compare what is said later regarding Acetone]. The urine which has this odor upon the addition of chlorid of iron sometimes gives a burgundy-red reaction ("chlorid-of-iron reaction," Gerhardt), which shows the presence of aceto-acetic acid.[3] Usually the odor of apples is even more noticeable in the breath of the patient than in the urine, and it may be noticed in the breath alone.

The apple-odor is observed in individual cases of *diabetes mellitus*. It especially occurs in diabetic coma or as the precursor of this condition, but it also exists—and, indeed, often for a long time—without the occurrence of coma.

Unusual odors may be imparted to the urine by medicines: after taking turpentine, violet odor; after cubebs and copaiva, the aromatic odor of these drugs.

Foul, albuminous urine, but especially urine that contains pus, develops, as the result of certain organisms, sulphuretted hydrogen—hydrothionic urine. Sometimes this fermentation, with the development of sulphuretted hydrogen, seems to take place in the bladder

[1] See above, under Reaction of Normal Urine, p. 364.

[2] See p. 360.

[3] See further, p. 407.

(cystitis). On the other hand, if the urine when first passed is clear, and upon being promptly examined is found to contain sulphuretted hydrogen, it is probable that there has been resorption of SH_2 into the blood or into the bladder from the intestine, or from a dépôt of pus in the neighborhood of the bladder; under which circumstances the general symptoms of poisoning have occasionally been observed.

Urinary Sediments.—We are to call to mind the sediments, previously mentioned, which may occur in normal urine. On the other hand, these same sediments may sometimes be observed as pathological signs, as is shown in what follows:

All formed constituents which separate when the urine is allowed to stand are reckoned as "sediments," whether they can be recognized with the naked eye or only under the microscope, or whether they are organized or are really "deposits." As previously mentioned, in order to examine the sediment it is desirable carefully to pour off from the vessel containing the urine the upper part; the lower turbid or already settled portion is to be put into a glass with a pointed bottom, and again allowed to settle. Then follows the examination with the naked eye and with the microscope. For the latter we take up some of the sediment with a pipette closed by one finger placed upon the upper end and introducing it to the bottom of the pointed glass, when it is to be opened again for a moment; then it is withdrawn and carefully wiped off and a drop of its contents allowed to flow upon an object-glass. [A slide with a depression in the center making a shallow cell is very convenient, since a larger drop can be examined at each time.] Upon this we place a glass cover, and examine it with a magnifying power of about 400 diameters. If the sediment is very scanty, we are to focus the microscope so as first to examine the edge of the covering-glass. It may happen that the sediment is so scanty that we cannot see anything at the bottom of the glass with the naked eye, but by carefully removing a drop from the bottom of the glass and placing it under the microscope we may possibly make out formed constituents, as a few casts (contracted kidney).

Stenbeck's Sedimentator[1] [Centrifuge].—This instrument is of extraordinary advantage for quickly separating all kinds of solid particles which are suspended in liquids. The principle is very simple: A portion of the liquid is centrifugated for a few minutes in small tubes similar to test-tubes. All the solid constituents are assembled at the bottom of the tube, which is somewhat narrowed—crystals, cells, red blood-corpuscles, micro-organisms, casts, etc. The method is applicable to all liquids.

It is necessary to color the urinary preparations only when examining for certain micro-organisms.[2]

Organic sediments (epithelia, white blood-corpuscles, casts) may be *stained with osmic acid*, which makes them visible, and also shows fatty degeneration if it exists. One or two drops of the sediment are put into a 1 per cent. solution of osmic acid, shaken, allowed to settle, and the sediment examined. The fat is stained black, brown-black, or gray.

[1] Instruments of this character are now made everywhere, and are readily obtainable.

[2] See p. 383 *ff.*

1. SEDIMENTS OF ORGANIC BODIES OR THEIR DIRECT PRODUCTS.

Mucus.—Physiologically, this exists only in small quantities.[1] It is increased in all diseases of the urinary passages, but especially in cystitis, and also in fever.

Some mucous forms are characteristic: in the form of minute roundish floccules, the size of a millet-seed or the head of a pin, they are tolerably characteristic of mild cystitis. Under the microscope they show white blood-corpuscles lying closely to one another, and they are apparently conglomerations of white corpuscles.

In the form of threads 1 to 2 cm. long—*gonorrheal threads*—which are sometimes more purely mucous in character, and, again, contain abundant pus-corpuscles: they occur in chronic gonorrhea or as the residuum of a past attack.

Finally, we find microscopical mucous threads, *cylindroids* (see Fig. 137, page 376), which may be confounded by the inexperienced with tube-casts. They are found in nephritis by the side of the casts in cystitis but also in health. They are distinguished from urinary casts by their usually being of considerable length, their mucus-thread texture, their very varying thickness (as fine as threads, especially at the end), and their tape-like appearance. The nature and significance of these formations have not yet been satisfactorily elucidated. They are found in diseases of the urinary passages, the kidneys being healthy, as in cystitis and pyelitis, but besides they occur in some cases of nephritis in company with casts. We have found them in slight acute hemorrhagic and non-hemorrhagic nephritis, and several times in greater numbers than the casts. At present we cannot regard as successful the attempt to classify these formations according to their renal or non-renal origin.

Chemical proof of mucus in solution: The addition of acetic acid makes a flocculent precipitate, which is not again dissolved by an excess of acid, nor is it again dissolved by heat, as is the case with a precipitate of urates produced by acetic acid.

In women mistakes may arise from the admixture of vaginal mucus with the urine.

Blood or Red Blood-corpuscles.—The appearance of the urine varies very remarkably in *hematuria.* Sometimes there is a considerable bloody sediment, not infrequently partly coagulated; again, only a fine deposit of red blood-corpuscles spread out evenly; and lastly, sometimes a more brown-red, clear, or dark-brownish sediment. The red blood-corpuscles may be so scanty as to escape detection with the naked eye. This distinction pertains to the amount of the blood, and its having been for a longer or shorter time in the urine—that is, with reference to the location of the hemorrhage. (Regarding the color of the urine, see page 368.)

Hematuria occurs—

(*a*) In diseases of the kidneys—that is to say, in acute and chronic hemorrhagic nephritis, in embolic hemorrhagic infarction of the kidney (valvular disease of the heart), in septic hemorrhage of the kidney (acute endocarditis), in marked engorgement of the kidney, with new formations, and, lastly, in injuries to the kidney.

[1] See p. 363.

(*b*) In certain diseases of the urinary passages, and also of the pelvis of the kidney (nephrolithiasis, tumors), of the bladder (severe cystitis, tumors, stone), of the urethra (gonorrhea), and with parasites of the urinary canal.[1]

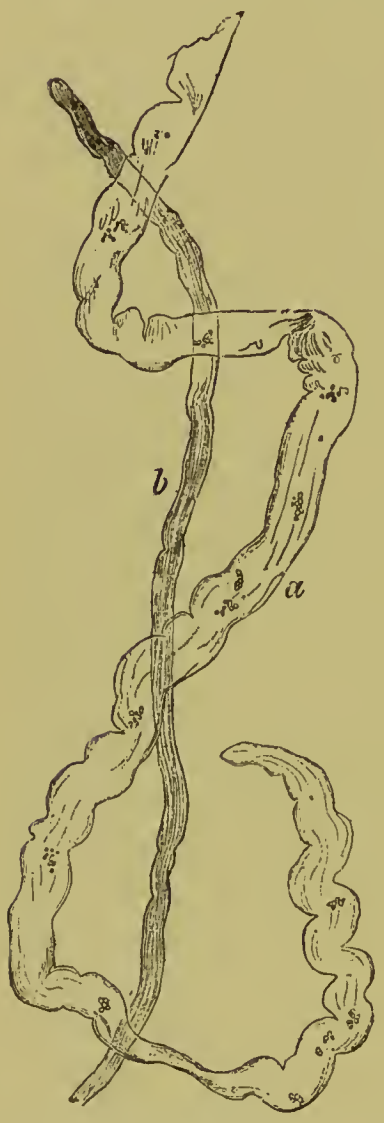


FIG. 137.—Cylindroids (see p. 375) (v. Jaksch).

Moreover, hematuria has *symptomatic significance* for recognizing diseases of other kinds. Thus it occurs in scorbutus, morbus Werlhofii, hemophilia, and, lastly, in the rare hemorrhages of the kidney or urinary tract that are due to leukemia.

From the appearance of the sediment and the way it is passed a conclusion with reference to the *location of the hemorrhage* and the kind of disease will be made from the following points of view:

A small amount of blood—or at least a not too abundant quantity of blood—uniformly mixed with the urine, the color of the blood being retained or, more frequently, changed into a brownish color, points to a hemorrhage of the kidney. That this is its source can be more certainly proved by the microscope showing blood-casts.[2] Where there is renal hemorrhage the blood-corpuscles are always more or less discolored, as rings or shadows. Cells and casts, if present, are stained brown by the coloring matter of the blood. A brown color of the sediment and of the urine indicates acute hemorrhagic nephritis. The sudden occurrence of bloody urine, with valvular disease of the heart, points to renal infarction. Individual red blood-corpuscles occur in very concentrated urine in renal engorgement.

In hemorrhage of the pelvis of the kidney, especially that caused by stone, the urine usually alternates between being bloody and free from blood, and this either because there are temporary hemorrhages or because the ureter of the diseased side is for the time being stopped, and then the urine that is passed only comes from the sound side. The blood may for a time escape very freely; in rare cases it may be passed in the form of vermiform coagula (casts of the ureter), which give great pain as they are passed.

Cystic hemorrhages, especially in villous tumors, may be so free as to be fatal. The urine is not intimately mixed with blood, especially if the patient lies quietly in bed; at first there is little or no blood at each urination; but then, again, pure blood is sometimes passed. On the other hand, in hemorrhage from the urethra blood comes only at

[1] See p. 382.

[2] See p. 382.

the beginning of the urination. Here sometimes there is an escape of blood between the urinations. Works upon surgery treat more at length of hemorrhages of the bladder and urethra.

Microscopical Examination.—In every respect this is the most valuable method for recognizing hematuria, especially from the following points of view: 1. Because the separate red blood-corpuscles can be discovered where neither the fluid portion of the urine nor the sediment shows the color of blood, and where, also, the fluid portion does not show the reaction of the blood-pigment.[1] 2. Because it alone establishes the differential diagnosis between hematuria and hemoglobinuria. 3. Because, from the condition of the red blood-corpuscles, from the presence of possible blood-casts,[2] we can sometimes determine that there is renal hemorrhage.

In hematuria we find more or less abundance of red corpuscles. In decided hemorrhage, especially from the lower portion of the urinary tract, these are only slightly changed. If retained for some time in the urine, and particularly if they are scanty, as in renal hemorrhage, they are smaller, have granular contents, or are more or less markedly discolored. If they are very pale, then we have the so-called rings. If there are no red blood-corpuscles in a urine that is bloody and certainly contains hemoglobin,[3] or if they are very scanty in a urine that contains a good deal of hemoglobin, then we have hemoglobinuria.[4]

Besides red blood-corpuscles, we frequently find in the sediment, according to the disease present, still other formed constituents; in cystitis, first of all, white blood-corpuscles, phosphate crystals; in nephritis, casts and white blood-corpuscles. A considerable amount of blood in the urine makes it somewhat albuminous.

With women we must remember the possibility of being deceived by the menstrual blood.

Hemoglobin.—In hemoglobinuria there is usually a brown or brown-black sediment, which consists of brown flakes and fine granular detritus. A few red blood-corpuscles are likewise found. If casts and epithelium are present, they are often colored brown.

Pus; White Blood-corpuscles.—It is rare that a considerable amount of pus is passed by the urethra. It sometimes appears in severe cystitis, and in the highest degree, when a neighboring dépôt of pus breaks into the urinary passages, either into the pelvis of the kidney or into the bladder. In the first instance, perinephritic abscess, and in the second pericystic, parametritic, perityphlitic abscesses, are the conditions which may occasionally manifest themselves by sudden pyuria. The pyuria may quickly pass away, but cystitis may follow.

Admixtures of pus—i. e. sediment of white blood-corpuscles of greater or less amount—may be caused by inflammation of the mucosa of the urinary passages; that is, by pyelitis, ureteritis, cystitis, urethritis, or by nephritis. In the latter case they are never very abundant.

Pus-sediment at first sight is yellowish, purulent, lighter, or wholly white in appearance. In pyelitis and cystitis, if the sediment is scanty, what is seen are mostly small distinct balls, smaller than millet-

[1] See p. 400.
[2] See Casts, p. 382.
[3] See Examination of the Dissolved Portion, p. 370.
[4] See this.

grains, which under the microscope are found to be globular conglomerates of pus-corpuscles. They are far too large to possibly come from the kidneys, and hence they point positively to the urethral passages.

In *nephritis* the pus-sediment is often finely powdered, loose, and may suggest a sediment of phosphates. This appears exceptionally also in chronic cystitis. In inflammation of the urinary passages the sediment generally becomes a peculiar compact jelly from the mucus it contains; in alkaline urine it is due to a mucus-like swelling of the white blood-corpuscles.[1]

The **microscopical examination** shows the white blood-corpuscles more or less changed according to their amount, the length of time they have been in the urine, and the reaction of the latter. In alkaline urine they are very clear and much swollen. Of the diseases of the kidneys, acute hemorrhagic nephritis and subchronic (chronic parenchymatous) nephritis produce a relatively abundant amount of pus-corpuscles. Senator says that among the pus-corpuscles of nephritis there are only a few eosinophile clear leukocytes, but leukocytes with one or more nuclei (those with one nucleus are often very much more numerous). The mononuclear cells resemble in part lymphocytes, but the body of the cell is large, so that they look like young epithelia.

To a slight degree pus makes the urine albuminous; a considerable amount of albumin in the urine is always due to renal albuminuria. When the quantity of albumin in the urine is slight, the question may arise whether we have nephritis either as a separate disease or as a complication of cystitis or pyelitis. This can only be answered by the infallible sign of nephritis—that is, casts in the urine.

Fat-drops.—The fat accompanying chyluria may, as was previously mentioned, exist in the urine as a sediment, but also as a cream-like or swimming layer or in the form of large drops. We must remember that it may be due to impurities, as the use of an oiled catheter, etc. The microscope shows minute particles of fat or large drops which markedly refract the light. In the first case the fatty character of the sediment may be most quickly recognized by the grease-spot formed upon paper by the sediment. We may also shake it up with ether, and then allow the ether to escape by evaporation.

The occurrence of fat-drops free and attached to casts, adipose, white blood-corpuscles, is very important in diagnosing *large white kidney*.

Epithelium.—We find in the urine the epithelium of the urinary passages and the epithelium of the renal urinary channels [urinary tubules]. In addition, in women very frequently, but especially when there is leukorrhea, we have flat epithelium from the vulva. The epithelial cells in transition are everywhere very similar. But renal epithelium is usually easily recognized as such.

While in normal urine only individual flat epithelial, and sometimes caudate, cells occur, in inflammation of the urinary passages we meet a large quantity of the three species of cells named. Usually they are well preserved. It is misleading to form a conclusion from the kind

[1] See above.

of cells as to the location of the inflammation (especially whether of the pelvis of the kidney or of the bladder). The vulva being excluded, a large quantity of flat epithelium points to the bladder. Abundant caudate, but especially overlapping, "tile-like," roundish cells with larger nuclei were formerly often regarded as characteristic of inflammation of the pelvis of the kidney; but more recently this view has come into discredit.

Renal epithelia occur in considerable numbers only in affections of the kidney, and especially in nephritis. If their form is well preserved, they are recognized without difficulty as polygonal or round-cornered cells of peculiarly sharp contour, with large oval nuclei and a decidedly granular, often yellowish-looking, protoplasm. They are small—not larger than white blood-corpuscles, sometimes smaller. In acute hemorrhagic nephritis they are often coarsely granular, brownish in color; in the large white (butter) kidney, but sometimes also in the first disease, we not infrequently see them in all stages of fatty degeneration.

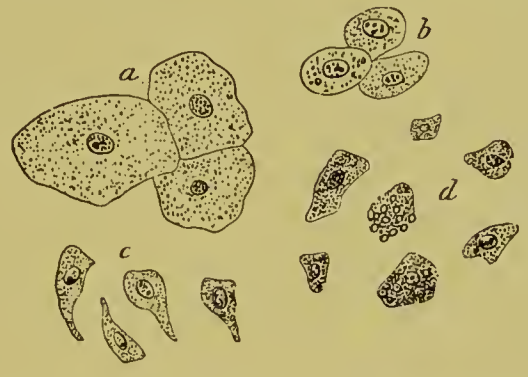


FIG. 138.—Epithelium from the urine.

a, *b*, epithelium from the bladder, from the pelvis of the kidney; *c*, caudate epithelium (pelvis of the kidney?); *d*, renal epithelium, partly changed into fat.

Regarding cylindrical epithelium, see under Casts.

Shreds of Tissue.—Shreds of connective tissue and "caseous crumbs" are found in tuberculosis of the urinary apparatus.

Particles of carcinomatous tissue are separated in carcinoma of the kidney, but have been more frequently observed in *carcinoma villosum* of the bladder. Only particles which distinctly show the structure of carcinomatous tissue are of importance here. Single, or also several pretended "cancer-cells" lying close to one another, have no diagnostic value.

Spermatozoa.—After every discharge of semen these are seen in the urine. Hence they are not unimportant for detecting masturbation. They also occur in spermatorrhea. Lastly, sometimes they are found after epileptic attacks; also now and then with severe diseases of all kinds, as in typhoid-fever patients.

Casts.—These important form-elements of the urine were discovered by Henle in 1842. As we have mentioned before, occasionally isolated casts may be found in the centrifugated urine of healthy persons without there being simultaneously the least trace of albumin chemically demonstrable. Casts have also been found in the albumin-

less urine of persons suffering from disturbances of circulation (Radomyski), also in icterus—particularly frequent, however, in tuberculous patients. Moreover, casts of a peculiar nature have been observed in the urine of diabetic patients before the beginning, or during the onset, of coma diabeticum (Külz and Aldehoff). But, apart from these particular cases, casts in the urine are a phenomenon accompanying *renal albuminuria.*

It is said that from the occurrence of icteric casts we may suspect the presence of biliary acids in the urine. They are intensely colored by biliary pigment.

We concern ourselves only with the occurrence of casts with albuminuria. By their presence these not only permit a conclusion that there is a disease of the kidneys which causes albuminuria, but by their *quantity* and *character* also enable us to diagnose the exact nature of the disease. Regarding their numbers, the casts are scanty, and then usually hyaline,[1] in engorgement of the kidneys, in fever, in physiological albuminuria; and, lastly, they are temporarily present in contracted and amyloid kidney. There is often here a sediment which is scarcely or not at all visible. In making a preparation we must, with the greatest care, take a few drops from the bottom of the urine-glass and examine the preparation with great thoroughness. If the *hyaline casts* are scanty, it greatly facilitates finding them to stain any that may be present by the addition of a drop of Lugol's solution or gentian-violet solution placed upon the edge of the cover-glass. Casts are very abundant in acute, and frequently also in chronic, nephritis. In these diseases they may form the principal portion of a tolerably abundant sediment.

Variation in the quantity of the casts is to be observed in all the diseases named. Sometimes, after a period of stagnation, it seems as if the casts are passed in greater abundance. This is not very rare in *amyloid nephritis,* also in attacks of *acute nephritis.*

In size and form the casts vary greatly. We will speak further regarding this.

As to their nature, we distinguish the following kinds of casts:

Hyaline Casts.—These are of great variety as to length and breadth; sometimes not so broad as a white blood-corpuscle (thin hyaline casts), and, again, five or six times as broad (thick or medium casts). In length they may be as much as 1 mm. They are homogeneous and clear as water, with a very fine outline, hence often very difficult to see; the ends look as if broken off, rounded, or even clubbed. For aggregation of substances within them, see below. They occur in company with other forms in all diseases of the kidney. Exclusively hyaline casts occur most frequently in contracted and amyloid kidney, also in fever and with [renal] engorgement.

A special kind of hyaline casts are the *waxy*, so named from their dull luster and usually yellowish color. Sometimes they show the amyloid reaction with iodin and iodid of potassium—brown, then violet with sulphuric acid. We cannot form a conclusion from them as to the nature of the disease of the kidney; certainly they are not pathognomonic of amyloid kidney.

[1] See below.

Additions to the hyaline and also to the waxy casts frequently occur in the form of red and white blood-corpuscles, renal epithelium, crystals, granular masses, which in turn may show urates, phosphates, albuminous or fat-granules, and, lastly, bacteria. Among these additions those of special significance are red blood-corpuscles, as in hemorrhagic nephritis, possibly adipose renal epithelia, white blood-corpuscles (granular spheres), and free fat-granules. These adipose elements, if abundant, are important for the diagnosis of large white or fatty kidney.

In some cases of pyelonephritis we have seen hyaline casts which were split like a pair of trousers. These might possibly have their origin in collective tubes (?).



FIG. 139.—Hyaline casts (narrow and tolerably broad ones).

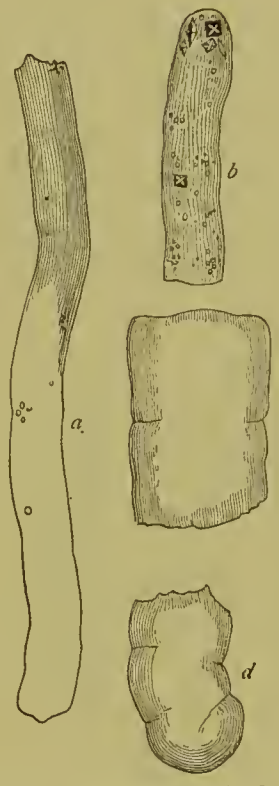


FIG. 140.—Waxy casts (v. Jaksch). *b*, a cast containing crystals of oxalate of lime.

Chemical Nature and Origin of Hyaline Casts.—Much has been written about the chemical nature and origin of hyaline casts. They must by no means be considered as a phenomenon inseparably accompanying albuminuria, as there is an albuminuria without hyaline casts (and without any other casts) and hyaline casts without albuminuria. It is principally this circumstance which has led to the assumption that all casts, and particularly hyaline ones, originate in the most diverse parts of the urinary ducts from the epithelia of these ducts. Several reasons, however, are opposed to this opinion, into which we cannot here enter. The most valuable investigations of recent times on the nature of these casts have been made by Ernst. He found that in acute as well as in chronic nephritis hyaline casts in part gave positive results with Weigert's method of fibrin-staining: some were wholly stained, some showed a stained nucleus and an unstained stratum lying concentrically around the nucleus; others, again, an unstained nucleus and a stained outer stratum; finally, some had taken up the staining substance in single spots. Ernst thinks it possible, although not yet proved, that hyaline casts are originally fibrinous, and are gradually converted into a hyaline-like substance which no longer reacts to the stain. It is not yet made clear from which part of the kidney the casts originate.

Granular Casts.—These are generally coarse or finely granular

hyaline casts, with additions to their contents, as above. But, especially in acute nephritis, conglomerate casts of albumin in lumps and granules also occur, sometimes stained or mixed with hematoidin.

Blood-casts are conglomerations of red blood-corpuscles held together by coagulation. They are important as indisputable signs of renal hematuria.

Epithelial casts are either hyaline casts with the addition of renal epithelium (recognized by their sharp outline and distinct large nuclei) or they are true epithelial tubes. In both cases they have the same significance—the free desquamation of renal epithelium, especially as it occurs with acute hemorrhagic nephritis.

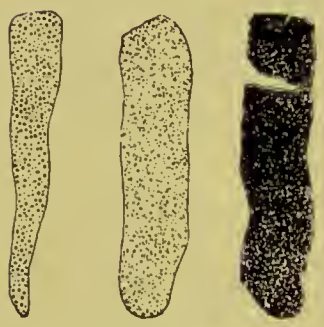


FIG. 141.—Granular casts (v. Jaksch).

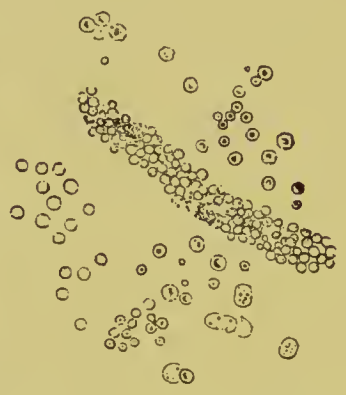


FIG. 142.—Red blood-corpuscles, partly as "rings" and cast of red blood-corpuscles (Eichhorst).



FIG. 143.—Epithelial cast (v. Jaksch).

Casts of lumps of hemoglobin in hemoglobinuria, urate casts in the newly-born (uric-acid infarction in connection with ammonium urate), and casts of bacteria in pyemia (?) are very rare occurrences.

We may confound casts with cylindroids,[1] also with threads of linen or other adventitious materials in the urine. Cleanliness and practice in examining guard one from mistake.

Animal Parasites.—Echinococcus.—Shreds from echinococcus bladders, scolices, and hooks are met with in the urine if an echinococcus of the kidney or from the neighborhood of the urinary apparatus breaks into the urinary passage. Urination is often attended with severe pain, especially by attacks of colic during its transit through the ureters. They may be preceded by anuria from obstruction of the urethra, obstruction of one ureter, and "reflex" suppression of secretion upon the sound side (or reflex spasm of the sphincter vesicæ).

Distoma hæmatobium, an exotic from Egypt, located in the roots of the portal vein, also particularly in the plexus vesicalis, causes hematuria. The eggs of the parasite make their appearance in the urine.

Strongylus gigas located in the pelvis of the kidney causes pyuria and hematuria.

Filaria sanguinis, an exotic from the East Indies, Japan, China, and Australia, located in the large lymph-vessels, among other things causes engorgement of the lymph-vessels of the bladder: chyluria

[1] See p. 375.

(and likewise galacturia[1]), and hematuria (peach-red urine). Besides, the urine contains embryo filaria, round-worms of delicate structure, lying in a fine sheath, with lively motion. In width they are about that of a red blood-corpuscle; in length, 2 to 3 mm.

Oxyuris vermicularis, trichomonas vaginalis (an infusorium) from the vagina, and, in one case under my observation, the larva of a fly, *musca vomitoria* (!), may become mixed with the urine.

Vegetable Parasites and Fungi.—Normal fresh urine, free from impurities, is not entirely free from fungi.[2] A number of bacilli and cocci colonize in urine that has been standing for some time, of which those of special interest are the ones which cause alkaline fermentation, changing the urea into carbonate of ammonia.[3]

In *cystitis* the findings seem to differ according to whether there is alkaline fermentation or not. In cystitis without alkaline fermentation of the urine a series of micro-organisms may be found which are to be regarded in part as the exciters of the disease. Among these the principal ones are the bacterium coli commune and the proteus. These bacteria are looked upon as the exciters of the inflammation of the mucosa.

The *alkaline fermentation* of the urine within the bladder which accompanies severe forms of cystitis, however, is produced by other micro-organisms if they enter the bladder. These are principally chain cocci (*micrococcus ureæ, micrococcus ureæ liquefaciens*), but also certain bacilli (a so-called *bacillus ureæ*, Leube and others). It is the presence of these fungi, in addition to the ammoniacal odor, which distinguishes simple alkaline urine from urine that is alkaline from fermentation.[4] If these schizomycetes are very abundant, they may form the greater portion of the sediment of the urine. Besides these, of course there are always pus-corpuscles and vesical epithelia, and also crystal forms of ammonio-magnesium phosphate and of the urate of ammonia.[5]

Alkaline fermentation of urine in the bladder always signifies a severe form of cystitis. It is principally found in cystitis following severe paralysis of the bladder and following the introduction of an unclean catheter.

In *pyelitis, cysto-pyelitis*, and *cysto-pyelonephritis* the bacterium coli commune seems to play the principal part, but besides this we must always bear in mind here tuberculosis and gonorrhea.

The certain demonstration of the bacterium coli commune can only be made by culture of urine received into sterilized vessels, and in women patients it must be drawn with the catheter.

Tubercle bacilli in the urine are an absolutely sure sign of ulcerating uro-genital tuberculosis. But in this disease, especially when there is tuberculosis of the pelvis of the kidney or of the kidney of only one side, the ureter of that side is temporarily or permanently stopped. If tubercle bacilli appear at all in the urine, they are generally abundant, not infrequently even in masses, and with an arrangement which reminds one of a pure culture. Fig. 144 exhibits an excessive development of this kind. In purulent urinary sediment tubercle bacilli can be demonstrated just as distinctly as in the sputum. In cases of persistent inflammation of the urinary tract it is well to examine for tubercle bacilli, so as

[1] See this. [2] See p. 361. [3] See p. 363. [4] See p. 373. [5] Compare p. 386.

not to overlook them. Tuberculosis of the uro-genital apparatus often gives rise to symptoms which for a long time simulate chronic gonorrhea, a simple leukorrhea, ordinary cystitis, pyelitis, hydronephrosis, hypertrophy of the prostate. The examination of the urine for tubercle bacilli should never be omitted when such conditions as have just been mentioned are associated with tuberculosis of other organs, as of the lungs, or with suspicious local phenomena (orchitis, chronic peritonitis,

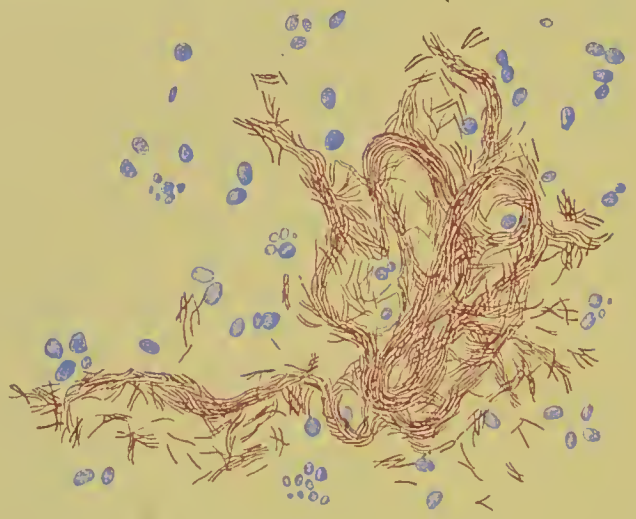


FIG. 144.—Pure culture of tubercle bacilli in the urine in tuberculosis of the genito-urinary apparatus. Zeiss's homogeneous immersion $\frac{1}{12}$, eye-piece No. 4. Drawn with a camera lucida; magnified about 1100. Author's observation.

etc.), or when there is anemia, emaciation, or hectic fever. Therefore, if there is decided anemia, wasting, and continued fever, as well as in cases of long-continued gleet, every purulent urinary sediment should be examined for tubercle bacillus.

Gonococci (Neisser) occur in the pus of recent gonorrhea in clusters, in epithelial cells, and in *pus-cells*. They are comparatively large cocci, 1.6 to 1.8μ long and 0.8 to 0.5μ broad. They most frequently appear as rather compact heaps of diplococci; sometimes an individual coccus is seen with a transverse band like a kind of roll. But the essential characteristics are, in the first place, their appearance in heaps in the white blood-corpuscles and then their discoloration by Gram's method. If stained after Gram and unstained, and then afterward stained again with Bismarck-brown, they even take up the latter color just the same as the cells do. Sometimes, particularly in the gonorrhea of women and in chronic gonorrhea of men also, there may be but extremely few gonococci, or they may be entirely absent for a time. In the latter case Neisser has made injections with slightly irritating substances, and thus produced a temporary appearance of the gonococcus in the secretion. Neisser recommends these stimulating injections as an important aid in diagnosis. It is best to use a watery solution of hydrargyri chloridum corrosivum 1 : 10,000. As yet, we have had no personal experience with this method. The gonococcus may easily be confounded with other similar cocci which occur in the urethral secretions in benign urethritis, and even in health. But it is settled now that the above-mentioned qualities belong only to the gonococcus (Neisser).

The gonococcus is to be stained with gentian-violet or methylene-blue or fuchsin, and then rinsed in water.

It must not be forgotten that acute and chronic purulent urethritis may be caused by other micro-organisms—streptococci, diplococci, and tubercle bacilli—and likewise that they may be free from bacteria.

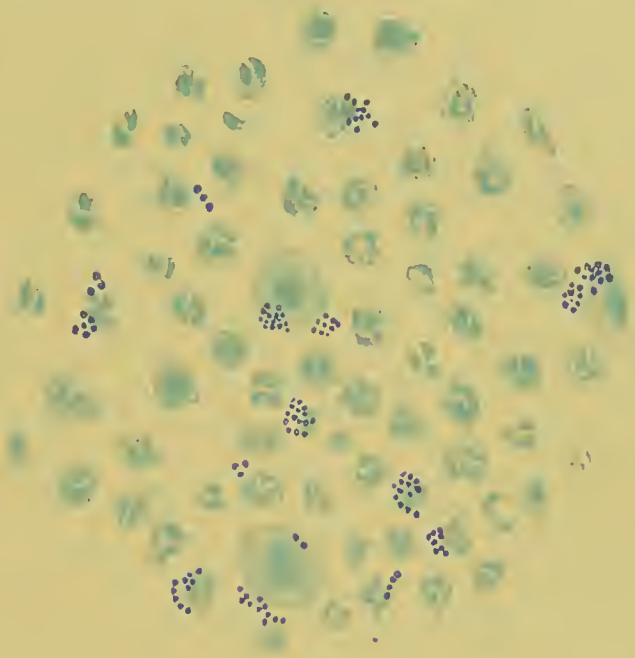


FIG. 145.—Gonococci in pus from urethra; dry preparation. Stain: methylene-blue.

Pathogenic fungi which circulate in the blood, in individual cases, are found in the urine: thus, tubercle bacilli in acute miliary tuberculosis, typhoid bacilli, equinia, erysipelas cocci in erysipelatous nephritis (Fehleisen), spirillum recurrens in complicating hemorrhage of the kidney (Kannenberg), pus-cocci in pyemia or endocarditis (Weichselbaum and others). Also, casts of micrococci are described in septic processes (Litten and others).

Lastly, in cases of *acute nephritis* bacteria have recently been found in the urine and in the kidney which have been regarded by different authors as the specific excitants of the nephritis. As yet, these cases are too much isolated and too uncertain to permit us to form a definite conclusion.

A small form of sarcina is found (rarely) in alkaline fermentation in the urine. It, as well as the other fungi named, is regarded as the cause of the transformation of the urea. Leptothrix buccalis occurs as a foreign substance, as from the preputial sac (Huber).

The occurrence of the yeast fungus, saccharomyces, in urine containing sugar is not unimportant. Here it causes acid fermentation. In urine that does not contain sugar some yeast-cells are found occasionally, but they do not increase.

2. INORGANIC SEDIMENTS.

A greater portion of the organic and inorganic combinations which normal and pathological urine contains may be precipitated from it and

appear as sediment. A distinction must generally be made whether such sediments appear in the still warm, recently passed urine, or whether the urine is clear when first passed and precipitates its constituents only when cool or after it has been standing for some time.

According to the investigations of Moritz, all the crystals of the more important substances precipitated from the urine contain an albumin-like organic frame-substance. Organic nuclei are also demonstrable in those concretions which are not distinctly crystalline, but are globular and the like, and even in the minute granules of sedimentum lateritium. This is true whether the precipitates have formed in the urinary passages or outside of the body, either spontaneously or by chemical additions.

Hitherto it has been supposed that only the coarser pathological concretions of urine contained such an organic nucleus, and from this it has been concluded that alterations of the mucosa in the urinary passages which furnish abundantly such nuclear substance gave the first impulse to a formation of concretions. This line of argument is no longer valid.

(*a*) THE MORE FREQUENT INORGANIC SEDIMENTS.

From acid urine there are deposited—*uric acid*, *uric-acid salts*, (*sodium*, *lime*), *oxalate of lime*.

From the faintly acid, neutral (amphoteric), alkaline urine there are deposited—*ammonio-magnesium phosphates*, *phosphate of lime*, *carbonate of lime*, *urate of ammonia*, and sometimes *uric acid*.

All these substances may occasionally be deposited from healthy urine.[1]

Uric Acid.—As is stated above, we find this as a deposit not only in acid, but sometimes in neutral and alkaline urine. It can often be



FIG. 146.—Uric acid and urates (Funke).

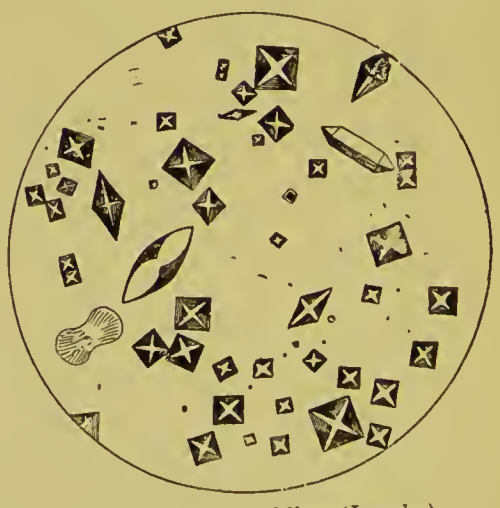


FIG. 147.—Oxalate of lime (Laache).

recognized with the naked eye in the form of yellowish-red, glittering granules, which are located upon the side of the urine-glass, or in the form of a yellowish-red powder at the bottom of the glass. Uric acid

[1] See p. 373.

deposited from the urine always has this yellowish-red color, while the chemically pure uric acid is colorless. Under the microscope it shows the greatest variety of crystal forms and crystalline figures (see Fig. 146). The basic form is the rhomboidal plate. But this is rare; more frequently we have derivatives of this, the so-called "whetstone" (with a cross or in druses), "barrel-shaped," also peculiar bundles of prisms; lastly, amorphous lumps and clubs with separate, shining, smooth surfaces,—all easily recognized by their distinct color. We may artificially produce a separation of uric-acid deposit by adding to the urine some concentrated solution of salt and allowing it to stand for twenty-four hours. Ordinarily, chemical reaction is not necessary.

New-born babies during the first days of life often excrete a rather large amount of uric-acid crystals in the freshly-passed urine, and their kidneys also at autopsies usually show a so-called uric-acid infarction. This hitherto unexplained phenomenon is physiological.

After the first days of life, however, uric-acid crystals in urine recently passed must always awaken a suspicion of uric-acid diathesis, while their presence in urine that has been standing for some time only admits the conclusion that it is not exactly wanting in uric acid, and nothing more.

Amorphous, roundish, gravel-like concretions in the urine are always pathological.

Urate of Soda and Lime.—When concentrated urine cools there is often a very abundant sediment, colored a flesh-red by the urinary pigment—"brick-dust sediment" or *sedimentum lateritium*. When cooled to zero C. we can obtain it from any urine. It will be most easily recognized by the fact that it immediately completely dissolves when the urine is warmed (not boiled, because then there is a phosphatic cloudiness and also coagulation of albumin,[1] if present). Under the microscope the urates of soda and of lime are seen as very fine grains. They incline to settle upon the casts, and especially upon mucous threads. Uric-acid crystals form about half an hour after the addition of some muriatic acid.

From concentrated urine the lateritious sediment is deposited at the ordinary temperature of the room, especially in engorgement of the kidneys, in attacks of diarrhea, in fever, and also in health.[2] We should never conclude from its presence that there is increased separation of uric acid. We can only determine this by ascertaining the amount of uric acid and urate separated in twenty-four hours.

A *sedimentum lateritium* in *fresh, still warm urine*, as well as coarser sediments of urates (sand, gravel), always gives rise to the suspicion of nephrolithiasis. This is especially the case if blood is found in the urine, or even if only a few red blood-corpuscles are found by the microscope in the urinary sediment.

In all cases of such concretions the *chemical proof of uric acid* must not be omitted. The concretion is powdered; some of the powder is mixed with a drop of nitric acid upon a porcelain spoon and slowly evaporated. A beautiful orange-red discoloration, which is turned into purple-red by the addition of ammonia, proves the presence of uric acid (*murexid test*).

Oxalate of Lime.—Single crystals of this may appear in any urine

[1] See p. 393.

[2] See p. 363.

that has been standing for some time. The crystals are almost always tolerably small, sometimes minute regular octahedra, which are conspicuous by their perfect form and strong refraction of light (envelope-



FIG. 148.—Bulbous forms of urate of ammonia; triple phosphates (Laache).



FIG. 149.—Phosphate of lime (Laache).

form). They are rarely hour-glass- and dumb-bell-shaped. The crystals are insoluble in water, and are thus distinguished from chlorid of sodium.



FIG. 150.—Ammonio-magnesium phosphates (after Meyer: *Semiologie des Harns*). *b*, tombstone crystals; *c*, crossed and feather forms.

These crystals occur in the urine in great abundance after eating certain fruits and vegetables, as apples, pears, cauliflower, and the different kinds of sorrel; and also in *diabetes mellitus*, *catarrhal icterus*,

hypochondria. Moreover, we cannot conclude, without further evidence than the mere occurrence of a somewhat large amount of these crystals, that there is increased separation of oxalic acid (*oxaluria*). The disease described by English physicians (and Cantani) as oxaluria does not seem to be a unity. This oxaluria occurs in *cachexiæ* (tuberculosis, cancer).

Ammonio-magnesium phosphate (triple phosphate) is found in urine that is simply alkaline and that is undergoing alkaline fermentation. Sometimes it forms the principal portion of the whitish sediment. The basic form is the rhombic prism; it is well formed in the "coffin-lid crystals," often also of various other forms, and is then more difficult to recognize. The triple phosphates are all perfectly colorless and soluble in acetic acid, thus contrasting with oxalate of lime.

Phosphate of lime as a *basic salt* occurs in amorphous grains in alkaline fermentation of the urine. It is soluble in acetic acid, but not by heat. As a *neutral salt* it occurs in simple alkaline urine in the form of long wedges or knife-blades. These disappear in alkaline fermentation.

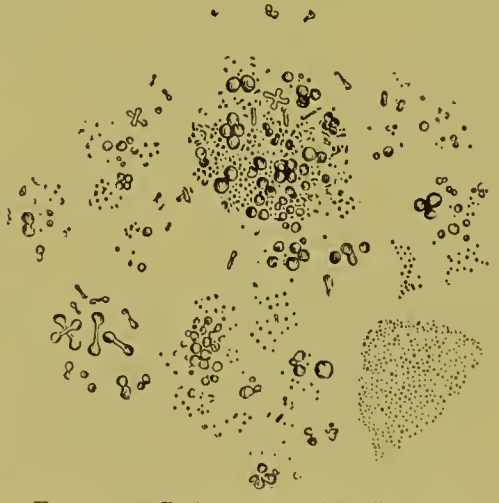


FIG. 151.—Carbonate of lime (Laache).

Carbonate of lime in the form of spherules or crossed drumsticks seldom occurs in alkaline urine. ["In highly alkaline urine, in which the alkalescence is caused by carbonate of ammonia set free by decomposition of urea, carbonate of lime occurs in small quantity, but in an amorphous form. This is the only form in which I have yet seen carbonate of lime in human urine." —Beale.] It is dissolved by the addition of muriatic acid, with effervescence.

The so-called **phosphaturia** is a condition in which phosphates and carbonates are precipitated before or immediately after the urine is passed. But there is no increase in the phosphoric acid. The precipitation is probably produced by the alkalinity of the urine. Phosphaturia occurs in neurasthenia, hypochondria, chronic articular rheumatism.

Urate of ammonia accompanies triple phosphate in alkaline fermentation. The characteristic form is that of the thorn-apple (grayish-yellow or brownish opaque balls, from which fine needles project). When muriatic acid is added uric-acid crystals develop under the cover-glass.

(*b*) MORE RARE INORGANIC SEDIMENTS.

Hematoidin is exceptionally found in the forms of needles and plates mentioned before (see Fig. 46). Sometimes we see white blood-corpuscles which contain hematoidin needles, which project through the cell-membrane.

Leucin and Tyrosin (see Fig. 152).—The characteristic forms of these substances, which almost always appear together, are sometimes



FIG. 152.—Leucin (*a*) and tyrosin (*b*), cholesterin (*c*), xanthin (*d*) (Meyer).

found in the sediment, more often only when we have evaporated the urine in a water-bath to the consistence of syrup or until we slowly boil down a drop of urine upon an object-glass until it is almost dry. Leucin appears in the form of faintly shining spheres, which sometimes, if large, show radiating lines and concentric rings. Tyrosin crystallizes in very fine needles, which commonly form druses and bundles.

Leucin and tyrosin are products of the decomposition of albumin. They do not occur in normal urine. Diseases in which they are found and for which they may have diagnostic value are *acute yellow atrophy of the liver* and *acute poisoning by phosphorus*. They are also seen in variola and typhus abdominalis [typhoid fever], as well as in pernicious anemia (Laache).

Cystin sometimes occurs in the urine in health. Large quantities of cystin in the urine may cause the formation of cystin-calculi and excite cystitis, and are thus a pathological condition in themselves. According to recent investigations (Baumann, Brieger), there seems to be a connection between the occurrence of ptomaïns and cystin in the urine. Brieger assumes that by the presence of certain ptomaïns in the intestinal canal (and sometimes in mycotic enteritis) cystin forms a combination with the ptomaïns in the intestine, which overflows into the urine. There the compound decomposes and cystin is again set free. Sometimes this does not take place, and so calculi are formed. The ptomaïns, in turn, may cause inflammation, especially cystitis. Cystin, besides occurring in the urine in the form of calculi, is seen in the form of extremely thin, six-sided, and very perfectly formed colorless plates.

(*c*) CONCRETIONS IN THE URINE.

Urinary concretions originate in the pelvis of the kidney or in the bladder. According to their size they are called sand, gravel, or stone. Concretions formed in the renal basin when passing through the ureter cause more or less violent attacks of pain (*renal colic*) proportionate to their size. Sand and gravel usually pass without pain. The coarser concretions which remain in the kidney or bladder at times cause characteristic pain.

Most frequently the concretions consist chiefly of uric acid and urates. They are then brown or brown-black, and tolerably smooth on the surface. Stones of oxalate of lime are densely hard and have a rough surface (mulberry calculi); they are dark brown. A combination of layers of uric acid and oxalate of lime is likewise met with. Phosphatic calculi are tolerably soft, but not infrequently they contain a kernel of the first-named substances (phosphate deposited upon the stone from the alkaline urine of cystitis [excited by the original stone]). Finally, we must mention stones of cystin and (extremely rare) xanthin. All these stones, with the exception of the phosphatic calculi, are formed in acid urine.

A simple *qualitative chemical examination* of such a concretion, instituted for the purpose of recognizing the substances most frequently occurring, is made in the following manner:

A sample of the substance is finely powdered, and then slowly raised to a red heat upon a porcelain spoon. If it does not burn at all or if only a very small portion of it burns, it consists mostly of inorganic combinations, most likely of oxalate of lime or of phosphates.

Another sample is dissolved in dilute hydrochloric acid: if it contains carbonic acid, it effervesces. Then filter, and mix a part of the filtrate with ammonia till its reaction becomes alkaline, after which add acetic acid till the reaction becomes slightly acid: a white precipitate, insoluble by heat, is oxalate of lime. If there is no precipitate after the addition of the acetic acid, add some acetate of uranium: a yellowish precipitate of phosphate of uranium indicates *phosphoric acid.*

If the substance is for the most part destroyed by red heat, a new sample is tested for uric acid by the murexid test.[1] If the murexid test gives a negative result, dissolve a portion of the powder in undiluted nitric acid in the bottom of a test-tube, and evaporate a drop of the solution slowly on a porcelain spoon: a lemon-colored residue, which is not altered by ammonia, but takes on a reddish hue on the addition of a solution of caustic potash, proves the presence of *xanthin.*

EXAMINATION OF THE URINARY CONSTITUENTS IN SOLUTION.

1. ANOMALIES IN THE QUANTITY OF THE NORMAL CONSTITUENTS.

In disease the normal constituents of the urine are variously increased or diminished. These quantitative variations, however, can only exceptionally be made use of for the diagnosis of disease. But they are important for determining the change of material and the removal of material that can be carried off by the urine in various diseases. This requires throughout an exact quantitative analysis, for the different

[1] See p. 387.

"approximative methods" have no value at all. We cannot here go into an explanation of the exact methods, but must refer to the hand-books upon urine-analysis. However, we mention briefly the most important anomalies which belong here. We have already mentioned the quantities of the normal constituents of the urine (page 365).

Urea.—This is increased in fever, either absolutely, as in pneumonia, or relatively—that is, in relation to diminution in the amount of food taken. It is also increased in diabetes. We find it diminished in all forms of nephritis, but especially in uremia; in cachexia of all kinds, especially if there is dropsy; and, lastly, sometimes in acute yellow atrophy of the liver. The very decided *increase* in the amount of excretion of urea which takes place immediately after the crisis in pneumonia is designated as *post-epicritical*. It is probably connected with the increase in the amount of water secreted by the kidney.

Sehrwald[1] devised a simplification of Knop-Hüfner's method of determining the amount of urea, which seems to us to be very practical and relatively exact. We have not yet had an opportunity to thoroughly test the method. At least we recommend that it be tried.

Uric acid is usually increased parallel with the urea in fever. Besides, it is increased in leukemia and pernicious anemia (with the first often very markedly), also in all diseases which affect the interchange of gases in the lungs; and, lastly, with the uric-acid or gouty diathesis apart from attacks of gout, during which it is often diminished.

The total amount of nitrogenous material in the urine, most important for determining the metamorphosis of tissues, approximately agrees with the amount estimated from the urea, because uric-acid, creatinin, and xanthin bodies are insignificant in amount compared with the urea. Besides, the most practicable method for the quantitative determination of the urea (Liebig's) is really a determination of the total amount of nitrogen expressed as urea (C. Voit, Salkowski, and Leube). When determining both nitrogen and urea, of course it must be done apart from any possible albumin—that is to say, the latter must first be removed.

Chlorid of sodium is pathologically increased during the resorption of transudations and exudations, and also in intermittent fever, from the destruction of red blood-corpuscles (Kast). It is diminished in fever, nephritis, and in many cachectic conditions. [In pneumonia, during the stage of exudation and until resolution begins, the chlorids are diminished or disappear from the urine. While the disappearance of the chlorids from the urine is not characteristic of this disease alone, it shows that exudation is still going on or that resolution has not yet commenced.]

Sulphuric acid interests us chiefly with reference to the associated ethylsulphuric acid (phenol-, indoxyl-sulphuric acids). It is found with increased separation of indican and carbolic acid. Regarding the former, see page 369. The latter occurs with the internal and external use of carbolic acid.

It has been found that the phosphates are diminished in rachitis, also in acute yellow atrophy of the liver. In nephritis they are not infrequently diminished.

[1] *München. med. Wochenschrift*, 1888, No. 46.

2. ABNORMAL CONSTITUENTS.

Albumin.—It has already been mentioned that the urine of healthy persons not infrequently gives certain albumin reactions, but that, according to more recent investigations, these albuminous substances are not the same as occur in renal albuminuria. Moreover, this albumin reaction of normal urine is usually produced by those substances which form the so-called *nubecula:* mucin-like substances, principally *mucin* and *nucleo-albumin.* These substances originate in the urinary passages, and may be increased in all affections of the urinary tract, especially in catarrh, but also in nephritis.

It is difficult to separate these substances chemically, and therefore they have recently been classed together under the name of "mucin-like substance of the urine." Their presence may give rise to the assumption of a renal albuminuria.

Since the mucin-like substance is partly precipitated as the urine cools, forming the *nubecula*, it is recommended in testing for albumin always to use cold urine which already shows the *nubecula*, and to be careful not to get any part of the *nubecula* into the test-tube. I have frequently found, in testing freshly evacuated urine during office hours, that it gave a distinct reaction of albumin, while the same urine after standing for some time proved to be free from albumin.

Nevertheless, also in cold urine that part of the mucin-like substance which remains dissolved may give a part of the albumin reactions. To avoid this source of error we add an excess of acetic acid to a sample of urine, and, if it is concentrated, dilute it with a little water. Cloudiness indicates mucin-like substance. It is best to clarify the sample by repeated filtration, and then to examine it for pathological albuminous substances.

For practical purposes we recommend the following method for all cases:

Mode of Procedure.—If we expect to find a very small quantity of albumin in a given specimen of urine, it is to be examined only after it has cooled. When the reaction of albumin is distinct this precaution is unnecessary. If we find in the cold urine a very slight reaction, this may still be due to the mucin-like substance. To exclude this we make the above-mentioned reaction with acetic acid. If it is positive, we try to remove the mucin-like substance by repeated filtering or make the heat and nitric-acid test, which precipitates only serum-albumin and serum-globulin, and while cooling precipitates the albumoses, and, at any rate, only a trace of mucin-like substance.

[The mucins, as stated above, interfere with the tests for serum-albumin. Of the various methods for getting rid of the mucin-like substance, the following one can be relied upon for getting rid entirely of mucin from mucous membrane, partly of that from bile and also of serum-globulin if that be present: To 1 ounce of urine add ½ ounce of distilled water; add 30 drops of a 25 per cent. solution of magnesium sulphate, then 30 drops of a 25 per cent. solution of sodium hydrate. Shake thoroughly and allow it to stand for ten minutes; then filter through a wet four-ply filter. Dilute the filtrate with one-fourth its volume of glacial acetic acid or one-half its volume of acetic acid;

boil; set aside for an hour; then filter through powdered talc and a wet four-ply filter-paper.]

The albuminous substances, which in the conditions reckoned as albuminuria in the narrow sense can be separated, are *serum-albumin* and *serum-globulin*. Their amount varies from a trace to ½ per cent., very exceptionally more. Generally, it remains below ½ per cent. The secretion of hemialbumose is very rare, and thus far has not been found to have special diagnostic significance. Of late we are not accustomed to regard *peptonuria* as albuminuria. It will be considered at the close of this chapter.

A *renal* or "*genuine*" *albuminuria* occurs: in all forms of *acute* and *chronic nephritis*, in *amyloid kidney*, in *engorgement of the kidneys;* in *hydremic conditions of the blood*, as anemia, leukemia; in *fever*, and in *acute poisoning:* in these two last-named cases, especially in the latter, there occur, besides, *all the transitions to nephritis:* lastly, *after epileptic attacks*, and with *apoplexy* (*transitory albuminuria*).

It is no longer doubtful that in all these cases we have to do exclusively, or at least chiefly, with an injury to the epithelium of the loops of the glomeruli or Malpighian corpuscles. Healthy epithelium holds back serum albumin; when the epithelium is diseased, filtration of albumin is no longer restrained.

Besides, there has recently been discovered a peculiar form of albuminuria which is distinguished from other forms by the absence of all pathological signs in the urine, especially of cylinders—*cyclic albuminuria*.[1]

Further, albumin may appear in solution in the urine, originating in the urinary passages when blood and pus, as in cystitis, are mingled with the urine in the bladder. The amount of albumin, however, is then always small.

Qualitative Tests for Albumin.—We select a few, from the great number of tests for albumin, which have the tolerably uniform approval of authors,[2] and which, according to our experience, have the preference.

The preliminary condition is that the urine be not contaminated, as by menses, leucorrhea or spermatic fluid, and that it be clear. The latter is the more necessary in proportion as the amount of albumin is small. In order to be able to discover it when only a very little is present it is necessary to filter the urine until it is perfectly clear.

(*a*) **Addition of Acetic Acid and Potassium Ferrocyanid.**—By acetic acid the urine is rendered distinctly acid, and then the cold urine is mixed with a few drops of a watery solution of potassium ferrocyanid. There occurs a very fine flocculent precipitation of albuminous substances, serum-albumin, globulin, and also of the albumoses, but not of the peptones. A milky cloudiness is produced by the precipitate if the quantity of albumin be moderate. If the percentage of albumin be very small, the turbidness appears somewhat later, after about a minute. If any develops later still, it has no significance. The test is very safe and very sharp, but has the one drawback that it indicates also the mucin-like substance. Therefore it is recommended to note

[1] Regarding this, see p. 396.

[2] See, regarding them, Penzoldt's *Aeltere und neuere Hornproben.*

whether cloudiness appears after the addition of acetic acid. If there be acetic-acid cloudiness, it is made only a little more pronounced by the addition of potassium ferrocyanid, and shows that the mucin-like substance is present with absence of renal albumin. Then make other albumin-tests, especially that by boiling and addition of nitric acid, or precipitate in a new test the mucin-like substance with an excess of acetic acid, clarify by repeated filtration, and then examine the filtrate for albumin. In this case it is well to try to filter off the precipitate produced by the acetic acid.

The potassium ferrocyanid test will be rendered more sharp if it is compared with another test-tube which contains urine to which only acetic acid has been added. The reaction is made still more distinct by following the suggestion made by v. Jaksch, which is to superimpose the urine with a mixture of moderately concentrated acetic acid and a few drops of solution of potassium ferrocyanid. With a minimal quantity of albumin there is seen a whitish ring at the line of contact of the liquids.

(*b*) **Boiling and the Addition of Nitric Acid.**—If the urine has a neutral or alkaline reaction, acetic acid, diluted one to ten, must be added to render it acid before boiling. If there is cloudiness, it can only be due to one of two causes: albumin or phosphates. To determine which of these it is, we add about ten drops of nitric acid, when the phosphatic deposit is immediately dissolved; but if the deposit is albumin, this is made more distinct. When the albumin is somewhat abundant, the deposit can be immediately recognized by its floccular appearance. This test may also be made by mixing the urine with about one-fifth part of nitric acid and then boiling. The test is a sharp one, showing even 0.005 to 0.01 per cent. of albumin, and, being tolerably certain, in general is to be recommended. A second modification is perhaps somewhat more distinct than the first: there are precipitated immediately serum-albumin and globulin, soon after cooling the albumoses, and besides, probably, also a part of the mucin-like substance. A precipitation that takes place later than a quarter of an hour after cooling cannot with certainty be regarded as due to albuminous substances.

(*c*) **Picric-acid Test.**—We add to the urine a few drops of a concentrated watery solution of picric acid: if it immediately becomes cloudy, it shows albumin, but cloudiness appearing later shows nothing (Johnson, Penzoldt). There is precipitation of albuminous substances, including albuminous and mucin-like substances, and also resinous acids. Otherwise it is a certain and sharp test, not less to be recommended than the others.

Portable Tests for Albumin.—These are such reagents as the physician may easily carry with him and use at the houses of his patients. They are not to be strongly recommended, as they all have some shortcomings. There are two which give safe results if there be well-developed albuminuria:

(*d*) **Geisler's Albumin Test-papers.**[1]—These consist of a piece of filter-paper saturated with a concentrated solution of citric acid, and of another saturated with a 3 per cent. solution of potassium iodid,

[1 They may be obtained of several manufacturing chemists.]

added to a 12 or 15 per cent. solution of corrosive sublimate. We first put one of the strips of the first into the urine—if very alkaline, more than one—then one of the second papers, and shake it. Cloudiness due to albumin appears pretty promptly. Peptone is also precipitated, which in many cases can cause deception.[1] In concentrated urine urates are also precipitated, but these can afterward be dissolved by heat. Deception from the solution of particles of paper making a cloudiness is not possible if it is carefully examined. As a preliminary test at the sick-bed this method is to be recommended. But we ought not to be satisfied with its result, and should always afterward employ one of the tests previously mentioned.

(*e*) **Furbringer's Reaction.**—This consists of gelatin capsules containing mercuric chlorid, sodium chlorid, and citric acid. The reaction has about the same shortcomings as the former, only it is a little cleaner because minute fibers of paper are excluded.

If we examine the urine a number of times in twenty-four hours, and find that there is a periodic presence and absence of albumin, we designate this condition as *cyclic albuminuria*.[2] It never occurs after rest at night; the albumin is generally separated after exertion. In case this condition is suspected we are to examine the urine several times during the day, and especially toward evening, as well as directly after rising in the morning. Klemperer has made a very clear demonstration of the course of the separation of the albumin. He places about 5 c.cm. of the urine, passed at different times during the day, in a series of reagent-glasses, and then boils them with the addition of nitric acid. The height of the deposit in the glasses as they are arranged in a row, may be regarded as a direct delineation of the "albumin curve."

Behind these cases of cyclic albuminuria there are hidden, on the one hand, cases of disappearing acute nephritis, and, on the other, beginning chronic nephritis. Some of them, especially those which occur in children, must be regarded as benign "*functional*" (?) diseases. The differential diagnosis between functional albuminuria and nephritis is chiefly founded upon the question whether other nephritic alterations of the urine are present, particularly organic constituents, and what the condition of the heart is. Moreover, cyclic albuminuria not infrequently occurs in diseases of the heart, preponderately in infancy.

Some of the benign cyclic albuminurias, particularly those that occur after exertion, are nucleo-albumins.

Quantitative Test for Albumin.—Here, as in all quantitative determinations, the urine of exactly twenty-four hours must be mixed, and a portion from this mixture examined. The urine for exactly twenty-four hours can be obtained if we have the patient urinate early, say shortly before seven o'clock, and then keep all the urine that is passed after that hour till the next morning at exactly the same hour, passing his urine again at seven o'clock.

It is possible to make an exact quantitative determination only by

[1] See Peptonuria, p. 398.

[2] [In the *British Medical Journal*, Jan. 31, 1891, p. 218, Dr. Herringham gives a valuable and careful study of a case of cyclical albuminuria which was under his care at the West London Hospital.—*Translator.*]

completely separating the albumin from a measured quantity of urine. Filter, wash the residue upon the filter-paper, dry, and weigh it. (For particulars regarding these processes, see text-books upon Urine-Analysis.) This examination can only be conducted in a laboratory. There is no mode of procedure which is more simple, nor one that is so nearly exact as this. The polarizing method is only applicable when there is a considerable amount of albumin.

A substitute for the exact quantitative determination is quite commonly found by endeavoring to estimate the amount of deposit which results from the qualitative determination, especially by the boiling nitric-acid test; we wait a long time—till it has settled in the reagent-glass—and then we speak of one-half, one-quarter, or of the "whole" being albumin by comparing the volume of albumin that can be seen with the whole amount of urine in the reagent-glass. It may be assumed that one-half the volume of albumin, if the reagent-glass has stood for one hour, corresponds to about 0.2 to 0.6. This estimate is extremely unreliable, being chiefly dependent upon the size and thickness of the flakes of albumin. But if we always employ the same test for albumin, it is certainly not valueless for judging of the variations in the separation of albumin in the course of the disease.



FIG. 153. — Esbach's albuminometer.

More exact is the method with **Esbach's albuminometer,** although it acts upon the same principle—that is, on the determination of the albumin from the volume of the precipitate—and so is only approximative. Its greater exactness only rests upon the fact that to a given quantity of urine there is always added the same amount of a reagent adapted for uniform sedimentation and mixed in the prescribed way, that a definite period of time is allowed for sedimentation, and that the height of the sediment is measured.

This albuminometer—a graduated thick reagent-glass—is filled with urine to the mark *U;* from there to *R*, with the reagent. This reagent consists of 10 grams of picric acid and 20 grams of citric acid to 1000 of distilled water.[1] The glass is then closed with a rubber cork, turned upside down ten times, and allowed to stand undisturbed for twenty-four hours, best in a special stand. After this period of time we notice at what mark of the scale on the glass the albuminous deposit stands. The marks each give $\frac{1}{10}$ per cent. of albumin. As the scale only goes as far as 0.7 per cent., urine that is strongly albuminous must be diluted in a definite way before the test. We must avoid producing air-bubbles, because these cause the precipitate or a part of it to swim, and for this reason we are not to shake the glass. If there are air-bubbles, they must be removed with a pipette.

In most cases the method is sufficiently exact for clinical purposes.

[1] The exact amounts of both acids (chemically pure and dry) are to be dissolved in 1000 grams of water, made hot, and, after cooling, any deficit in the amount of fluid is to be made up by the addition of water to 1000 grams.

[The apparatus is not at all expensive, and can be obtained from dealers in chemical apparatus.]

A series of experiments, recently published by apothecary Dr. Rössler (Baden-Baden), gives the following differences:

	Albumin determined by weighing.	Albumin determined by Esbach's method.
1.	0.01535 per cent.	0.010 per cent.
2.	0.0286 "	0.04 "
3.	0.0515 "	0.03 "
4.	0.1271 "	0.07 "
5.	0.217 "	0.22 "
6.	0.328 "	0.28 "
7.	0.404 "	0.23 "
8.	0.483 "	0.36 "
9.	0.486 "	0.59 "
10.	0.66 "	0.33 "

From this table it is seen that the inexactness of Esbach's method is regularly too much upon one side, and then too little upon the other. Without visible cause, in some cases the precipitate is so small that after a few hours one is tempted to abandon the test.

Notwithstanding these defects, the small, very cheap apparatus is to be strongly recommended, because it is a great advance upon the simple estimate in the ordinary test-tube, and because we have no more exact method which anywhere nearly approaches this in simplicity.

Rare Forms of Albumin.—Peptone (v. Jaksch, Maixner, and others).—This never occurs in healthy urine. Pathologically, it occurs sometimes in ordinary albuminuria, and again independently—*peptonuria*. It occurs in a great number of very different conditions—in large abscesses, in emphysema, sometimes in pneumonia; likewise in acute rheumatism, scorbutus, phosphorus-poisoning; also, in carcinoma ventriculi, in puerperal fever, in typhus abdominalis [typhoid fever], etc. Hence this very remarkable substance has no value for diagnosis. Its determination, even qualitative (biuret reaction), is, for various reasons, difficult.

Albumoses do not occur in the urine very frequently (*"albumosuria"*). We shall refer to these urinary constituents very briefly, only remarking that we may suspect their presence if the urine becomes turbid as it cools after the use of the heat and nitric-acid test. Hitherto this substance has had no diagnostic significance.

Kahler has recently observed hemialbumose in multiple primary lympho-sarcoma of the spinal cord.

Fibrin occurs in the urine in *hematuria*, in deep-seated inflammation of the urinary passages, in tuberculosis, in poisoning with cantharides, and in chyluria. It is recognized by the fact that it coagulates spontaneously in the urine, although sometimes only after the urine has stood for some time. The coagula are then to be further examined.

In this place are to be mentioned two phenomena that occur in those diseases of the kidney that stand in close relation to albuminuria—*dropsy* and *uremia*.

The dropsy of kidney-disease manifests itself, very frequently, first in the skin of the face, especially at the eyelids. With contracted kidney

the edema is very fugitive, often changing its place; in a large number of cases it is entirely wanting during the entire course of the disease. With large white kidney it is more decided and stable; there is often a very soft, doughy edema. In this respect acute nephritis varies very much. In all forms of Bright's disease, from its association with heart-weakness, a new factor may come into play for the development or increase of the edema and effusion into the cavities of the body (dropsy of engorgement).

With reference to the cause of the dropsy in kidney-disease, no doubt the most important element is the diminished elimination of water by the kidneys. This retention of water often, especially if excessive, has the effect that even a slight, perhaps a scarcely noticeable, dropsy of the skin and subcutaneous tissue considerably disturbs the excretion of water by perspiration. At any rate, it is certain that the dropsy of kidney-disease is, in many cases, not explained by the retention of water; but neither is Cohnheim's hypothesis, that the walls of the vessels are abnormally previous, at all generally accepted. This whole matter is still an open question.

Uremia is an association of nervous manifestations which, at least in the majority of cases, is dependent upon the retention in the blood of urinary products (especially uric acid). In individual cases of "uremic" manifestations, however, this explanation is not correct, and the nature of such cases is not yet clear (edema of the brain, Traube (?)); sometimes anatomical changes in the brain, Strümpell (?), etc. We coincide with Strümpell's view, that uremia is a multifarious manifestation—a number of conditions which, by their presence and their phenomena seem to belong together, are in reality different.

Slight uremic symptoms may last, with slight changes, for weeks, even months, as somnolence, restlessness, headache, malaise, vomiting, dyspnea (uremic asthma), indications of Cheyne-Stokes' respiration, slight transitory disturbances of vision. The more *severe symptoms* are—decided cloudiness of intelligence, even to coma or delirium; maniacal conditions; convulsions, varying from single convulsive movements to pronounced epileptic attacks; and temporary amaurosis. There may be slowness of the pulse, with acceleration later, and fever. In individual cases there occur evident symptoms of cerebral congestion—convulsions, paresthesiæ, paralysis of an arm or of one side of the body, and aphasic manifestations.

Mucin and Nucleo-albumin.—These bodies have already been mentioned a number of times. They are products of the cells of the mucosa of the urinary tract, are present in all specimens of urine, and are precipitated in part as nubecula. They are increased in all forms of catarrh of the urinary tract, and also in nephritis. Their proof has been given on page 393.

Coloring Matter of the Blood.—The occurrence of this body has also been previously mentioned.[1] Here we have to refer to the *tests for hemoglobin or hematin in solution.*

First, it must be mentioned that of course the urine shows the presence of albumin in both hematuria and hemoglobinuria. The amount of albumin is always small, provided there is no albuminuria besides.

[1] See p. 370.

Blood-pigment will be shown to be present by the following procedures:

(*a*) *Heller's Test.*—A portion of urine is made decidedly alkaline with caustic potash and boiled in a reagent-glass: the phosphates are precipitated as very delicate floccules, which look like mucus and slowly sink to the bottom. They accompany the blood-pigment, and hence look brown or red-yellow. When the urine is concentrated we dilute it, after boiling, by filling the reagent-glass with water, because the color of the floccules is easily concealed. Urine that is poor in phosphates, as in nephritis, gives no phosphatic deposit. Such urine must be mixed before making the test with some that has the normal amount of phosphates. The color described as belonging to the phosphatic deposit occurs nowhere else, except with urine containing chrysophanic acid, but this latter is recognized by its change in color after the reaction. This test is very simple, certain, and with clear urine is tolerably distinct.

(*b*) *Test with Tincture of Guaiac.*—The reagent consists of tinct. guaiac, ol. terebinth. ozonisat., *āā.* 10 parts. A small portion of this, placed in a reagent-glass, is carefully covered with urine: when the coloring matter of the blood is present there is, besides the dirty, white deposit of resin, an indigo-blue ring. When shaken up the whole contents of the glass become a non-transparent, bright blue. The test is a very distinct one.

(*c*) *Test for Hemin.*—This is made with a large drop of urine or urinary sediment, exactly in the same way as has been described already [1] for finding it in the material vomited. The test is more distinct than the preceding, particularly if we boil it down in a porcelain dish and then apply the reaction.

(*d*) *Spectroscopic Examination.*—This gives the absorption-bands of methemoglobin—namely, in yellow, green, and red. Of course this is an extremely distinct test.

Indican.—(See page 369.)

BILE-PIGMENTS AND BILE-ACIDS.

Gmelin's Test for Bile-pigments.—We pour a small quantity of nitric acid into a reagent-glass, and add to it one or two drops of fuming nitric acid, forming a trace of an admixture of nitrous acid. To this mixture we very cautiously add a layer of urine, by permitting it to flow from a pipette down the side of the glass held obliquely. When the bile-pigment is abundant, if the fluids are kept carefully distinct there is a ring of green (blue), violet, and red. The first named constitutes the test. There is no reaction when there is only a small amount of bile-pigment.

Rosenbach's modification is decidedly more distinct. Filter some urine, not too little (about 200 c.cm.), through a medium-sized filter, unfold it, and place it upon a white surface, and then pour upon it a few drops of the mixture of nitric and nitrous acids. The colored rings form upon the filtrate.

Gmelin's test is still sharper if, after acidulating the urine with

[1] See p. 330.

acetic acid, we shake it up with chloroform, pour off the urine, and then with the chloroform, colored yellow by the bile-pigment, make a layer with the nitric-acid mixture.

Penzoldt recommends a filtrate prepared as in the Gmelin-Rosenbach test (allowing a good deal of urine to flow through), over which acetic acid is poured, and this is allowed to flow into a broad glass vessel, so as to have it in a shallow, but broad, layer. The acetic acid becomes yellow-green, gradually becomes green (quicker, if it is warmed), even bluish-green. Penzoldt declares that this test is very distinct.

Rosin's Test.—H. Rosin has recently recommended a test with tincture of iodin (tinct. iodin, 1 part, absolute alcohol, 9 parts). The reagent is carefully superimposed on the urine to be examined. If bile-pigment is present at the junction of the liquids, there is a green ring. The test is very beautiful, but we have not yet tested its sharpness and certainty.

Pettenkofer's Test for Bile-acids (glycocholic, taurocholic, and cholal acids).—This test is based upon the fact that the addition of a weak solution of cane-sugar (1 : 500) and a trace of concentrated sulphuric acid to urine causes a violet-red color. We must be careful not to have the resulting elevation of temperature too high—at most not higher than about 50° C.

For various reasons this last reaction is uncertain. Its result is reliable only when the bile-acids, if present, have been isolated. At any rate, the bile-acids have only a slight diagnostic value: a trace sometimes occurs in normal urine, while we find in undoubted cases of jaundice due to engorgement of bile often none, or only a trace, because frequently in the transmission it becomes broken up in the blood. Hence in cases of icterus we cannot account for the absence of the bile-acids from the urine by the assumption that it is not an hepatogenous icterus. On the other hand, an abundance of bile-acids in the urine proves that the jaundice is due to engorgement of bile. Moreover, it is clear that if we wish to logically explain "hepatogenous" icterus by the idea of engorgement of bile in the liver, we must assume an increase of the bile-acids in this jaundice also. As a matter of fact, this is found to be the case in toxic "hemato-hepatogenous" icterus (arseniuretted hydrogen, toluylendiamin—Stadelmann).

Grape-sugar.—Pathologically, grape-sugar occurs in the urine—

1. In *diabetes mellitus*, usually in considerable quantity—as much as 2 to 5 per cent. (minimum $\frac{1}{2}$, maximum about 10 per cent.). The urine is increased in amount, is bright and clear, of higher specific gravity, as has already been mentioned.

We will not here enter upon the differential diagnosis of the different forms of diabetes.[1]

2. As *pathological glycosuria* (Frerichs), usually in small quantity and almost always temporarily: *after poisoning* with carbonic oxid, curare, amyl nitrite, turpentine; sometimes with mercury, morphia, chloral, prussic acid, sulphuric acid, alcohol; again, *in acute infectious*

[1] See special papers, particularly those of Naunyn: "Die diätet. Behandlung des Diabetes mellitus," *Volkmann's Sammlung klin. Vorträge*, Nos. 349 and 350; also various handbooks.

diseases (typhoid, scarlet fever, diphtheria, etc.); *in diseases of the oblongata* (but here it is more lasting); and *from other neurotic causes*, as excessive mental exertion, neuralgia, injuries to the central nervous system, concussion of the brain, etc.; also, after *epileptic convulsions* and *apoplexia cerebri;* as *alimentary glycosuria*, after partaking of a considerable quantity of sugar,[1] or even after eating carbohydrates. The latter must always awaken a strong suspicion of commencing diabetes.

The *boundary-line between physiological and pathological alimentary glycosuria* is difficult to draw. The latter form depends upon a morbid disturbance, and more particularly represents the precursor of diabetes. According to Moritz, 100 grams of sugar will not produce any glycosuria in a healthy person, and he accords with the advice of v. Noorden to give 100 grams of grape-sugar on an empty stomach in the morning as a test for pathological glycosuria. If sugar appears in the urine, v. Noorden thinks commencing diabetes should be suspected. Such a test may be made now and again in members of diabetic families and in fat people.

It is to be remarked that the urine is always to be examined for sugar when it has a decidedly high specific gravity; but particularly if it is clear and abundant and, at the same time, has a high specific gravity.

After a single examination it is often difficult to make a distinction whether the case is one of diabetes or glycosuria. Only careful further observation of the patient can settle the question. Glycosuria is temporary and ceases definitely. Every return must direct the attention to a diagnosis of diabetes if one of the causes of glycosuria mentioned does not again come into play.

Qualitative Tests for Sugar.—**Bismuth Test** (with Nylander's modification).—For this purpose we employ Nylander's reagent: 2 parts basic bismuth nitrate and 4 parts sodium tartrate to 100 parts of 8 per cent. solution of sodium hydrate. Of this we take 1 part to 10 of urine and boil them together. After a few minutes, if there is only a little sugar—sometimes only after it has cooled—it becomes black from the reduction of the contents of the reagent-glass with the formation of the bismuth oxid if the urine contains as much as 1 per cent. of sugar.

It is evident that this is a very distinct test. It is only not applicable when there is albumin in the urine or ammoniacal fermentation has taken place. (Remove the albumin and apply Trommer's test.) Where there is only a slight reaction, it has no great certainty. Only a negative result shows that there is certainly no sugar (Kistermann).

Trommer's Test.—To a given quantity of urine we add about one-third as much liq. potassæ, and to this, drop by drop, of a 10 per cent. solution of copper sulphate, as long as it is held in solution by mixing. It is important to add, as exactly as possible, just so much of the copper sulphate as is dissolved by shaking; and hence the addition of the copper must be interrupted as soon as the first trace of a flocky precipitate remains after shaking the test-tube. Then it is heated, but not allowed to boil. An abundant precipitate of yellowish-

[1] See p. 366.

red hydrated cupric suboxid, which appears even before the fluid reaches the boiling-point, shows the presence of sugar. The yellow color of the liquid or a precipitate that takes place later may be caused by a very small amount of sugar, but also by uric acid and creatinin.

Albumin and salts of ammonia prevent the reaction, and therefore albumin must first be removed by boiling after acidulation with acetic acid. Urine which has undergone alkaline fermentation, however, cannot be used. Urine containing a large percentage of creatinin and uric acid sometimes prevents a positive result of the test if the percentage of sugar is small, as these substances are capable of holding in solution only a small amount of oxydulated[1] copper. It is necessary to make the fermentation test[2] with the urine of these cases.

When the percentage of sugar is considerable (over 0.5 per cent.), Trommer's test is very useful; below 0.5 per cent. it is not safe or distinct. If it gives a very small precipitate, or one that appears somewhat late, or if it results in a yellow coloration without precipitation, Nylander's test at least must be applied, but it is better still to use the fermentation test.

Phenyl-hydracin Test (v. Jaksch).—About 2 grains of muriate of phenyl-hydracin and 3 of sodium acetate are put into a reagent-glass which is filled half-full of water. After heating the glass is to be filled with the urine to be tested. It is allowed to stand for fifteen or twenty minutes in boiling water; then it is put into a beaker-glass filled with cold water. When there is a large amount of sugar there is formed a macroscopically visible deposit. With a small amount of sugar, after standing, there is a deposit, which can be seen with the microscope, of yellow needles, single and in druses—phenyl-glucosazon. Yellow plates and brown balls prove nothing. Albumin that may be present must previously be removed by boiling the urine. Von Jaksch urges this test because it is a very exact one. Its difficulty consists in this, that the needles of phenyl-glucosazon are sometimes not alike clearly characteristic in distinction from the yellow plates, etc.; which proves nothing, these latter not being crystallizable in alcohol. Nevertheless, the test seems to be a very sharp one, but its certainty is doubted by some.

Of the other very numerous tests for sugar we only mention the following:

Moore's liq. potassæ and boiling test, which causes urine that contains sugar to become brown—not a very certain and sharp test; and the *test with diazo-benzol-sulphuric acid and potash* recommended by Penzoldt.

One test, of great importance and highly recommended on account of its absolute certainty, is somewhat troublesome:

Fermentation Test.—This rests upon the peculiarity that yeast has of separating sugar into alcohol and carbonic acid (succinic acid, etc.). This test should always be applied when the reduction tests yield slight or doubtful results, and particularly when there exists a doubt whether the reduction is caused by sugar or so-called reducing substances.[3] It is especially necessary to take into consideration that these reducing substances which are not sugar (creatinin, uric acid,

[1 Copper oxydule = Cu_2O.] [2 See below.] [3 See p. 366.]

and others) may be present in increased quantity in fever and when the urine is concentrated. (According to Moritz, maximum reduction = 0.5 per cent. of grape-sugar.) After chloroform narcosis Kast has found, besides, a reducing substance in the urine which is probably a paired glycuronic acid.

The fermentation test may be made in a simple way, as follows: Three perfectly clean reagent-glasses are filled about two-thirds full of mercury. The first is then to be filled with some of the urine to be tested and a little yeast; the second is to be filled with normal urine and some yeast; the third with a thin, watery solution of sugar and yeast. It is well to add to each a drop of a solution of tartaric acid. All three tubes are now placed upside down in a tray of mercury by covering the openings with the thumb as we invert them. The second tube should not show any development of carbonic acid, but if it should do so the yeast was not perfectly free from sugar, and the experiment must be repeated with yeast that is perfectly pure. The third glass should show the development of carbonic acid, otherwise the yeast has become inactive. If the second and third test-tubes fulfil the conditions named, we may draw a safe conclusion from the behavior of the first tube: if it shows formation of carbonic acid, the urine contains sugar, but if it does not show development of CO_2, no sugar is present. The development of carbonic acid is recognized by the existence of gas in the upper part of the inverted tube. Its presence is made certain by its being absorbed when potash lye is introduced into the tube.

Fermentation-tubes are very helpful in employing the fermentation test (see Salkowski-Leube, Penzoldt). [Dr. Max Einhorn's fermentation saccharometer is one of the simplest yet devised. It is graduated so as to show the percentage of sugar.]

Quantitative Determination of Sugar.—This is indispensable if a case of diabetes is to be carefully observed, particularly for determining its severity, its course, and especially the effect of treatment. From the qualitative examination we cannot draw satisfactory conclusions as to the amount of sugar except by a comparison of the specific gravity of the urine with its quantity.

We make use of the mixed urine that is passed in exactly twenty-four hours.[1]

We only adduce two of the most important and most used methods —namely, that of estimating it with copper sulphate and by circumpolarization. The method recently recommended of determining the sugar by measuring the specific gravity before and after fermentation is very circumstantial, and requires more exact aräometers than those commonly used. The determination from the volume of carbonic acid resulting from fermentation (gaso-volumetric fermentation test) can only be carried out in the laboratory.

1. Estimating Amount of Sugar by Fehling's Solution (after Salkowski-Leube).—This method ascertains the percentage of the reducing substance in the urine; therefore, besides the percentage of grape-sugar, it measures also that of levulose—an inexactness, however, which is not of much moment. A considerable percentage of

[1] See p. 396.

albumin is disturbing, because then the precipitation of the suboxid of copper [copper oxydule] either does not take place at all or not promptly. If this is the case, the albumin must first be removed.

The principle is that in Trommer's test the copper oxid in an alkaline solution of grape-sugar is reduced to a lower state of oxidation: 5 parts of anhydrous grape-sugar will reduce 34,639 parts of pure copper sulphate to protoxid. The problem is to determine how much of a specimen of urine is necessary to reduce a certain amount of copper sulphate.

Solution I.: 34,639 grams of pure copper sulphate are, by warming, dissolved in about 100 grams of water, and the solution is then diluted to 500 c.c. It is to be set away, well corked.

Solution II.: 173 parts of sodium tartrate and 100 parts of officinal solution of sodium hydrate of the specific gravity of 1034, dissolved in water to 500 parts. This is to be kept in a well-stoppered bottle, but it must not be allowed to become too stale.

Mode of Procedure.—Mix equal parts of I. and II. The mixture (Fehling's solution) must not, when boiled, separate any oxydule. Ten c.c. of the mixture and 40 c.c. of water are placed in a deep porcelain saucer or in a little glass bulb. Thoroughly mixing the urine of twenty-four hours, we take a portion of this and dilute it with 9 parts of water (urine 1, water 9), and with this we fill a burette. The mixture in the saucer is brought to the boiling-point, and into this the urine in the burette is allowed to flow: there occurs a separation of oxydule and oxydule hydrate, and the blue color of Fehling's solution disappears. The instant when the fluid (if we incline the saucer) first completely loses its blue color shows the completion of the reduction. We allow the amount of urine necessary to complete the reduction to flow from the burette.

Calculation.—Since 0.05 gram of grape-sugar reduces 10 c.c. of Fehling's solution, therefore the quantity of the mixture which has escaped from the burette contained 0.05 gram of grape-sugar. We represent that quantity of the mixture by q; then the mixture in the burette contains $\frac{0.05 \times 100}{q} = \frac{5}{q}$ per cent. of sugar. And, since the mixture of urine was diluted tenfold, the urine itself contains $\frac{5 \times 10}{q} = \frac{50}{q}$ per cent. sugar—that is, five times the amount diluted, divided by the quantity of the mixture in the burette that was used.

The dilution of the urine is to be varied according to the amount of sugar it contains, which is to be done in such a way that the mixture in the burette shall contain about 0.5 to 1 per cent. of sugar.

Schmiedeberg's Modification.—This is a modification of Fehling's solution which can be kept for a long time: a solution of $CuSO_4$ 34.64 in distilled water 200.0 is mixed with a solution of mannit 16.0 to distilled water 100.0.

When used, to this solution is added solution of sodium hydrate (1.145 specific gravity, 62.4 per cent. of NaHO) 480.0, and then water added to make 1000.0. Mode of procedure and calculation is the same as above.

A Method of Approximate Determination of Sugar.—For practical purposes the approximative method suggested by Duhomme is very useful. Its basis is the estimation by Fehling's solution, but requires two Limousin's drop-counters, one for 1 c.cm. and one for 2 c.cm. In the former is placed exactly 1 c.cm. of the urine to be tested, and the number of drops contained in this c.cm. of urine is counted. Then by using the second drop-counter, 2 c.cm. of freshly prepared Fehling's solution and an equal amount of water are put into a test-tube; heat to boiling, add 1 to 2 drops of urine; shake, again heat to boiling-point, add 1 to 2 drops more of urine, boil, and so continue, repeating the process till the liquid is completely discolored.

Calculation.—The number of drops of urine in 1 c.cm. divided by the number of drops used for the reduction, gives the percentage of sugar. For instance, 1 c.cm. of urine contains 20 drops. Thirty drops have been used: hence $\frac{20}{30}$ equals 0.7; that is, the urine contains 0.7 per cent. of sugar.

Urine which contains a very large percentage of sugar must be diluted.

2. Determining the Sugar by Circumpolarization.—This depends upon the property of sugar to turn the plane of polarization to the right. Recently, the method has come somewhat into discredit, or it has been shown to be exact only when we exclude oxybutyric acid and any levulose that may be present, both of which turn the plane of polarization to the left. Regarding complicated methods (complete fermentation, etc.), see hand-books upon Urine-Analysis.

We do not give a description of the method by polarization, as a description of its use always accompanies the different apparatus sold. (We recommend particularly the simple apparatus made by Zeiss.)

It must be remembered that urines containing albumin have to be de-albuminized (acidified by acetic acid and boiled), and those that are too dark have to be decolorized. The latter is done by adding basic acetate of lead and ammonia till an abundant precipitation takes place. The filtrate is then sufficiently decolorized for polarization.

OTHER SOLUBLE CONSTITUENTS OF THE URINE.

Levulose sometimes occurs in the urine, in addition to grape-sugar, in cases of diabetes mellitus. It gives the chemical reaction of the latter, and for this reason it cannot, without complicated methods, be recognized, chiefly on account of a striking difference between the quantitative determination by Fehling's solution on the one side and the polarizing apparatus on the other. Levulose turns the plane to the left, but we must be on guard with reference to oxybutyric acid, which also turns the plane to the left.

Lactose occurring in puerperal patients, *inosite* in diabetes insipidus and albuminuria, can only be demonstrated in the urine when they are isolated.

Lipuria, as has been already mentioned,[1] occurs in chyluria. It has in one instance (Ebstein) been found in pyonephrosis; small quantities of fat occur with large white kidney,[2] in poisoning by phosphorus

[1] See p. 371.

[2] See p. 378.

and in diabetes mellitus, and also in health after taking very much fat, as cod-liver oil. The proof is by mixing it with ether. *Lapaciduria* (fugitive fatty acids in the urine) has recently been much studied, but thus far, from the standpoint of diagnosis, without significance.

Diaceturia, resulting from acetöacetic acid in the urine (v. Jaksch), never occurs under physiological conditions. It is observed in diabetes, and especially in the severe forms, which then sometimes end in coma; also in fever and as an independent disease (v. Jaksch); and both are apt to occur in children. Acetone[1] at the same time is always abundantly demonstrable in the urine.

Diaceturia, especially if it occurs in adults, generally indicates a serious condition. It is of considerable importance for judging of the severity of diabetes. Gerhardt's chlorid-of-iron reaction,[2] which is characteristic of diaceturia, although not perfectly reliable, is significant for the severe forms of diabetes. If diaceturia is present, oxybutyric acid[3] is also present in the urine. But it is important to observe that the chlorid-of-iron reaction may fail, and yet oxybutyric acid be abundantly present in the urine.

In diabetic coma, which to-day is almost universally regarded as an auto-intoxication by oxybutyric acid (Stadelmann, Minkowski), the chlorid-of-iron reaction is usually very distinctly present. Pronounced chlorid-of-iron reaction is also frequently a warning precursor of diabetic coma. It is to be remarked, however, that chlorid-of-iron reaction (and odor of acetone) may exist for a long time in diabetic patients without there ever being coma, and, on the contrary, coma may occur without chlorid-of-iron reaction ever having been present.

Von Jaksch supposes that the convulsions of children, so frequent in acute diseases, are always accompanied by diaceturia.

Proof of diacetic acid is made by *Gerhardt's chlorid-of-iron reaction.* Some solution of chlorid of iron is added, drop by drop, to the urine; sometimes there occurs an abundant precipitate of phosphates, which must be removed by filtration; then more iron chlorid must be added. If diacetic acid is present, the urine becomes a burgundy-red to deep dark-brown. The reaction is not entirely positive. If we wish to make certain we must observe the following directions: The test must be repeated with urine that has been boiled. Further, a portion of urine must be mixed with sulphuric acid, extracted with ether, and then the test repeated with the extract; lastly, it must be examined for acetone.[4] Diaceturia is certainly present if, in the presence of the chlorid-of-iron reaction of fresh urine, 1, the boiled urine shows no or only a slight chlorid-of-iron reaction; 2, if the ether extract shows a chlorid-of-iron reaction which fades in the course of twenty-four hours at the longest; 3, if acetone is present at the same time (v. Jaksch).

Oxybutyric Acid (β-oxybutyric Acid).—This acid has acquired an extraordinary importance in the pathology of diabetes, since we know that it is found in the urine in the severe forms of the disease (Külz, Minkowski); that its presence and quantity in these cases are about parallel with the severity of the disease or to the excretion of sugar (Wolpe); and, finally, that in diabetic coma it is without exception found in very great quantities in the urine. It is scarcely any longer

[1] See p. 409. [2] See below. [3] See below. [4] See below, p. 409.

doubtful that diabetic coma represents an auto-intoxication by the acid —an acid-intoxication (Stadelmann) in which the acid produces a poisonous effect by the absorption of alkali from the blood. In addition to diabetes, this acid has also been found in acute exanthemata, in scurvy, and in the state of starvation (abstinent lunatics).

In order to understand the frequent but not regular cöappearance of oxybutyric acid, of diaceturia, and of acetonuria, which will be mentioned below, it is important to remember that diacetic acid is a product of oxidation of oxybutyric acid, and, further, that diacetic acid easily decomposes into acetone and carbonic acid.

β-oxybutyric acid turns the plane of polarization to the left, and its presence in diabetic urine becomes probable when, after the sugar has been removed by fermentation, the urine turns the plane of polarization to the left. It is necessary, however, to see that no albumin is present. Albumin must be removed, if any is present, by boiling the urine after acidulation with acetic acid. Sometimes it is also necessary to decolorize the urine.[1]

The more exact qualitative and quantitative determination is difficult, and does not belong to the province of this work.

In close connection with the excretion of acid (Hallervorden, Stadelmann) stands the excretion of a substance with which the organism neutralizes the pernicious acids so long as it is able to do so —the excretion of ammonia.

[The Translator adds here a summary of Stadelmann's observations upon "Diabetic Coma," as given in the *American Journal of the Medical Sciences*, taken from *Deutsch. med. Wochenschrift*, 1889, No. 46:

"1. Diabetic coma, apart from accidental coma due to other causes, occurs only in the case of diabetic patients whose urine contains oxybutyric acids.

"2. Almost equivalent in value with the recognition of oxybutyric acid is the determination of the amount of ammonia in the urine, while it is also far easier of performance.

"3. Diabetic patients with an excretion of ammonia of more than $1\frac{1}{10}$ grams per day, are in danger of becoming severe cases of the disease.

"4. Patients excreting 2, 4, 6, and more grams of ammonia daily need constant watching by the physician, and are in constant danger of passing into diabetic coma.

"5. If the determination of the presence of oxybutyric acid or the estimation of the amount of ammonia cannot be carried out, at least the chlorid-of-iron test should be made. If this gives a more positive reaction, oxybutyric acid is present in the urine, and the cases answer to the statements made in the third and fourth conclusions. The converse of this, however, is not always true, for there are cases of diabetes with oxybutyric acid in the urine, and even suffering from diabetic coma, the urine of which does not give the chlorid-of-iron reaction."]

Ammonia in the Urine.—In normal urine it amounts to only about 0.5 to 0.8 *pro die*, but when oxybutyric acid appears it is increased proportionately. We must omit here the quantitative determination of this substance.

[1] See p. 406.

Acetone.—This is a product of normal, and of increased, decomposition of albumin, but traces of it are found in the urine in health. By *acetonuria* we understand an increase of this substance, and its presence is made known by the odor of acetone[1] if the excretion is abundant. Acetonuria is found in diabetes, sometimes in connection with diaceturia and excretion of oxybutyric acid, and hence sometimes in coma or as a precursor of it. However, abundant acetone also occurs without these other substances, and by itself it is without special significance. Moreover, it is found in gastric and intestinal disturbances, in inanition, in carcinoma (also without inanition), and in psychoses. But there also seems, especially in gastro-intestinal disturbances, to occur an auto-intoxication from acetone in connection with acetonuria (v. Jaksch), which intoxication is accompanied by epileptiform spasms and other phenomena of cerebral irritation, or also by signs of depression, which in the cases hitherto observed have ended in recovery. Also in these cases acetone appeared in the urine without diacetic acid, while on the other hand, as has been mentioned above, diaceturia seems never to occur without simultaneous acetonuria.

The exact test is complicated. Several methods have been given, which, if one wishes to be certain, it is best to employ simultaneously: 1. Distil the urine with some phosphoric acid. Several cubic centimeters of this distillate are mixed with a few drops of solution of iodin and potassium iodid: an immediate precipitate of iodoform-crystals proves the presence of acetone (Lieben). 2. We add to the urine some freshly-prepared mercuric oxid, obtained by mixing an alcoholic solution of potash with mercuric chlorid. Filter it, and cover the filtrate with ammonium sulphate: a black ring of sulphate of mercury shows acetone (Reynolds). Legal (cited by v. Jaksch) has devised a test for acetone which is a useful preliminary one: Several cubic centimeters of urine are treated with a few drops of a concentrated solution of sodium nitro-prusside and somewhat concentrated liquor potassæ. If acetone be present, a bright-red color is seen, which quickly fades, but upon the addition of some acetic acid changes to purple or violet-red.

Regarding the occurrence in the urine of *paired sulphuric acids* or of the products of their decomposition (here also belong indican, which has been previously mentioned, indoxylsulphuric acid), also of ptomaïns, ferments (especially pepsin), see the various special works upon these subjects.

Ehrlich's Diazo-reaction.—Some years ago Ehrlich discovered that by means of diazo combinations aromatic bodies could be demonstrated in the urine. The chemical nature of these substances is still unknown. They may be regarded as products of decomposition which appear only in certain conditions and are excreted with the urine. The reaction which takes place in the presence of such substances he calls diazo-reaction.

Mode of Procedure.—The reagents must be freshly prepared each time, and consist of the following:

1. 250.0 of highly diluted hydrochloric acid, saturated with sulfanilic acid (HCl 50.0; aq. destillat. 1000; sulfanilic acid 5.0).

[1] See p. 373.

2. 5.0 of a 0.5 per cent. solution of sodium nitrite.

The mixture may be very well made by putting 3 grams of No. 1 in a test-tube and adding 1 drop of No. 2.

Next, mix equal parts of urine and the reagent, and add somewhat quickly (not drop by drop) an excess of ammonia (about one-eighth volume). If the mixture assumes a red color, the diazo-reaction is present. The reaction occurs in different degrees, from light pink to deep red. The foam which forms from shaking shows the color very distinctly. Healthy urine, without exception, after this treatment takes a brown-yellow color.

In diseases without fever the diazo-reaction is only very seldom observed; and the same is true of chronic severe cachectic conditions of the most varied character. Here it has no diagnostic value.

But among the diseases with fever there are some to which it particularly applies. For example, it is almost always present in typhoid fever, except in very light cases. It occurs with typhus exanthematicus and in measles. It would therefore be of weight in deciding a doubtful case of typhoid fever, were it not also frequent in another disease which has often to be considered in doubtful cases of typhoid fever—namely, in acute miliary tuberculosis. For the diagnosis of recurring typhoid fever a positive result of the test has some decided value, particularly in differentiating it from other feverish complications of convalescence from typhoid fever, in which it is always absent, as in intestinal catarrh. Moreover, the reaction is also frequent in severe florid phthisis.

A prognostic value of the diazo-reaction obtains in typhoid fever, where its disappearance seems to signify a favorable result, and in phthisis, where its appearance is ominous.

THE URINE AS AFFECTED BY MEDICINES AND POISONS.

The determination as to whether a medicine has been taken or not may often be of diagnostic importance. A number of medicines may be directly detected in the urine: to those not easily or not at all demonstrable, according to Penzoldt's recommendation of a particular case, we add one easily demonstrable, even if given in small amount. The one most available for the purpose is an iodin salt (about 0.1 to 0.2 iodid of potash). If we find in the urine the reaction of demonstrable medicines that have been given, then we can naturally assume that any other which was mixed with it has been taken.

The urine has only a limited significance for the detection of poisons —in the first place, because they are excreted to so limited an extent as to show only a trace, or are not given off in a form to be detected.

We give here a few short directions:

Preparations of Iodin.—Add a couple of drops of red fuming nitric acid and about one-quarter as much chloroform as there is of urine; shake it gently; the chloroform gradually settles colored reddish-violet.

Bromin.—The same method; chloroform colors it brown-yellow.

Salicylic Acid.—The urine is made a blue-violet by the chlorid of iron (not burgundy-red).[1] When the amount of salicylic acid is

[1] See Diaceturia, p. 407.

small, we shake up the urine (to which some sulphuric acid has been added) with ether, and then apply the test.

Rhubarb and Senna.—See page 371.

Carbolic Acid, Naphthalin, Resorcin, etc.—The urine contains hydrochinon, and after standing for a time becomes olive-green to brown-black, even black. Exact determination requires peculiar methods.

Salol.—Urine containing this, as well as carbolic acid, becomes green to black, and at the same time responds to the tests for salicylic acid.

Antifebrin.—Add one-fourth volume of a concentrated solution of hydrochloric acid in a reagent-glass; boil for a few minutes; cool; add a few c.c. of a 3 per cent. solution of carbolic acid and a drop of dilute solution of chromic acid. The mixture becomes red; after the addition of ammonia up to an alkaline reaction, a beautiful blue (after Müller).

Antipyrin, Thallin.—Red coloration with chlorid of iron; moreover, thallin urine is green-brown.

Of the **poisons,** properly so called, arsenic, antimony, and lead, they pass off by the urine in very small amounts. This is true also of mercury and silver, but the former after long administration may be demonstrated without difficulty. We refer to the text-books upon toxicology. Of the *alkaloids*, quinia and strychnia are excreted in part unaltered; morphin also sometimes, but sometimes it is absent altogether. Also here we must refer to works devoted exclusively to toxicology.

EXAMINATION OF THE SECRETIONS OF THE MALE GENITO-URINARY APPARATUS.

Although with reference to the examination of the genital organs themselves we refer to the text-books upon surgery and sexual diseases, as may be seen from the superscription, we confine ourselves here to a consideration of the character of the *secretions* so far as this has a bearing upon diagnosis.

The **normal seminal fluid** is a mixture of the secretions of the testicles, the vesiculæ seminales, the prostate, and the glandulæ Cowperi (Fürbringer). It is a ropy, viscid mixture of mostly fluid and some compact substances resembling swollen groats. It is whitish in color, of a neutral to weakly-alkaline reaction, and has a peculiar characteristic odor. Microscopically, it contains large quantities of spermatozoa, some finely granulated testicle-cells of different size, and roundish grains, so-called prostatic granules. If some of the spermatic fluid be dried, there are formed the so-called sperma-crystals, large, light, oblong crystals, which are a product of the prostatic epithelia, as Fürbringer has shown. They have a certain superficial resemblance to Charcot's crystals, which we have met with in sputum, in feces, in leukemic blood, and in other places. According to exact crystallographic examinations, however, they are not identical with these (Cohn).

The sperma-crystals may be demonstrated in a particularly beau-

tiful manner by adding to the sperma upon an object-slide a drop of a 1 per cent. solution of ammonium phosphate and examining at the edge of the cover-glass after it has stood for several hours.

According to our present knowledge, the chemical composition of the sperma does not come into consideration for the purposes of diagnosis.

It is of interest to distinguish the individual constituents of the sperma according to their different origin:

1. The pure secretion of the testicles, according to examinations made in animals, is a homogeneous, viscid, whitish liquid which can be drawn out in threads. It furnishes the spermatozoa and testicle-cells of the sperma. The spermatozoa show a very lively individual movement in the fresh sperma.

2. The unmixed secretion of the seminal vesicles is gelatinous, slightly yellow, resembles swollen sago-grains, and furnishes the grit-like constituent of the sperma. When the sperma is not quite fresh these granules quickly dissolve.

3. The normal secretion of the prostate may be obtained by a digital compression of the gland through the rectum. It is a thin fluid, made milky-turbid by peculiar microscopic granules and stratified amyloid bodies. When mixed on a glass slide with a drop of a 1 per cent. solution of ammonium phosphate, and then evaporated, it forms the sperma-crystals.[1]

The **abnormal character of the spermatic fluid** has nothing to do with a diagnosis of *impotentia coëundi*—that is, as regards inability to cohabitate—for this condition occurs quite independently without any abnormality of the sperma; and, on the other hand, anomalies, and even entire absence of sperma, by no means excludes the ability to cohabitate.

It is necessary to distinguish—

1. **Aspermatism.**—This is a condition where during ejaculation there is no discharge whatever from the urethra. It is very rare, and is generally caused by stricture of the urethra or the openings of the ductus ejaculatorii. In stricture of the urethra the sperma flows backward into the bladder, later is forced through the stricture with the urine. Or the sperma may not pass during erection, but after the penis has become relaxed—that is, after cohabitation. The nature and location of the stricture must be ascertained by a careful local examination, possibly by means of an endoscope (Nitze). The most common conditions are: stricture from gonorrhea, diseases of the prostate, anomalies of the location and form of the colliculus seminalis. Moreover aspermatism has been observed with spinal diseases.

The so-called *temporary, relative aspermatism* (temporary absence of ejaculation, dependent upon sympathy, inclination for certain women) is considered by Fürbringer and Güterbock as related to, or identical with, psychical impotence.

2. **Azoöspermia.**—There is seminal fluid, but it contains no spermatozoa. This is much the more frequent form of sterility. The spermatic fluid nevertheless has the characteristic odor, very often it is in all other respects similar to normal, lacking only in spermatozoa.

[1] See preceding page.

There may be no other possible anomaly of the genital apparatus or possible disturbance of the potentia coëundi. This is true of the majority of cases.

Azoöspermia mostly depends upon obliteration of the seminal ducts, especially from epididymitis or funiculitis duplex as the result of gonorrhea. Moreover, it may be due to other severe diseases of the testicle—syphilis, tuberculosis, malignant tumors, congenital rudimentary development, etc.

There have been some cases of *temporary azoöspermia* observed; it may sometimes happen after very excessive abusus sexualis, and hence a single observation of absence of spermatozoa must be taken into account with caution.

Immobility of the spermatozoa in a fresh ejaculation or their deformity cannot, according to our present knowledge, with certainty be used for the diagnosis upon one or the other side.

Method of Determining Aspermatism or Azoöspermia.—The best way is to require the use of a condom in cohabitation. The contents of the condom should be examined as soon after the cohabitation as possible. It is conceivable that there are cases where the secretion cannot be obtained in this manner, but the other "method," closely approximating this, is not only disgusting, but, in our opinion, the results might not always be convincing. There are numerous difficulties in this connection, and for further details of this question we refer to Fürbringer's *Krankheiten der Harn-und Geschlechtsorgane.*

The conditions hitherto described can only be distinguished by microscopical examination. This is also the case where evacuations of sperm-like fluids from the urethra take place at other times than during coitus. We distinguish the following:

1. **Urethrorrhœa ex Libidine** (Fürbringer).—During erection, but without signs of ejaculation, a few drops of a liquid resembling egg-albumin, and containing only a few epithelia and round-cells, escape from the orifice of the urethra. Probably they are the secretion from Cowper's, possibly also of Littre's, glands. The condition is in itself without significance, but sometimes it is not easily distinguished from chronic gonorrhea.[1] Besides, urethrorrhœa ex libidine produces in the urine no forms similar to the gonorrheal threads, but its product floats in the urine as transparent jelly-like formations (Fürbringer).

2. **Prostatorrhea.**—This is an evacuation of normal prostatic secretion or of a thick fluid, mucous or muco-purulent discharge, sometimes constant, sometimes temporary, and again often passed during defecation or urination. The microscope shows pus-corpuscles, etc., as characteristic elements, amyloid bodies, and after evaporation, sometimes only after addition of ammonium phosphate,[2] Böttcher's crystals. Prostatic secretion sometimes causes threads in the urine similar to the gonorrheal filaments. Moreover, it is necessary to remember that chronic gonorrhea may exist at the same time. Prostatorrhea occurs with chronic prostatitis, both diffuse and suppurative.

Both with urethrorrhœa ex libidine and with prostatorrhea it is

[1] See this.

[2] See above.

possible that sometimes there may be admixture of individual spermatozoa.

3. **Spermatorrhea.**—The spermatic fluid may be passed without erection, most frequently during defecation, also at the close of urination or in walking, marching, climbing, etc. It may be perfectly normal or thinner than normal, or it may be mixed with pus or blood. These genuine pathological losses of semen occur in tabes and other spinal diseases; in epilepsy, insanity; in neurasthenic cases, especially those following excessive venery, but especially those due to masturbation.

The diagnosis of urogenital tuberculosis and gonorrhea has been treated of in the chapter on Urinary Apparatus, pages 383 and 384.

CHAPTER VIII.

EXAMINATION OF THE NERVOUS SYSTEM.

SCIENCE has not revealed in any organ of the human body an anatomical structure so fine and complicated as exists in the different parts of the nervous system. Nowhere else can be found tissues which differ so entirely and which are so clearly distinguishable clinically as here.

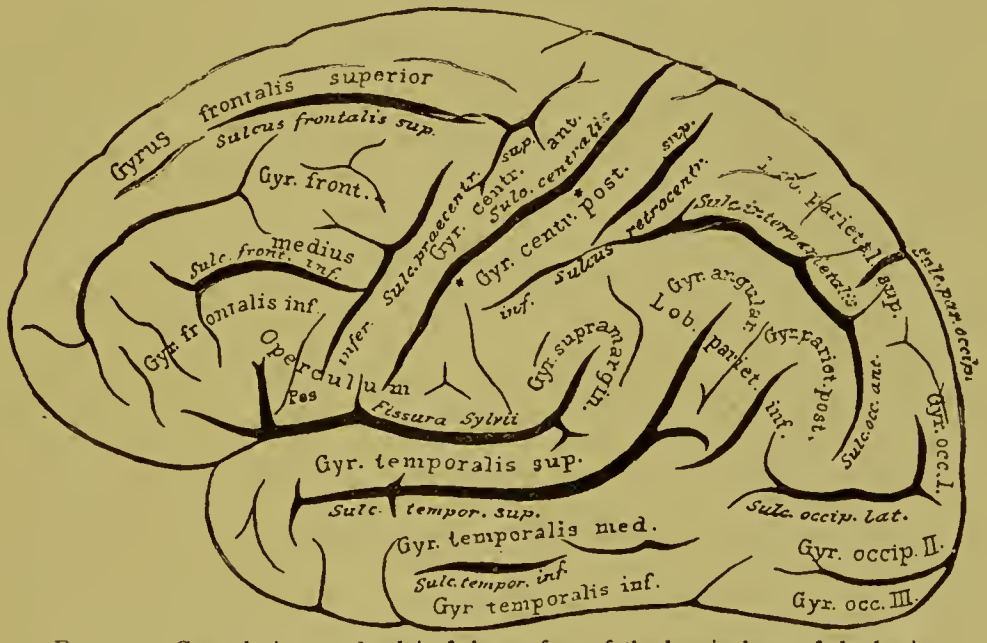


FIG. 154.—Convolutions and sulci of the surface of the hemisphere of the brain.

It is not easy to acquire a knowledge of its anatomy and physiology, but its study rewards the diagnostician in an extraordinary manner, for in no other domain of pathology can he with such certainty make a diagnosis which, on the one hand, is based on the anatomical relations, and, on the other, on loss or alteration of functions. To the purely scientific satisfaction of making a correct diagnosis is, in many cases, added the most important practical element, the discovery of means of cure. For, although hitherto some of the diseases of the nervous system have been little accessible to treatment, nevertheless, there are some in which everything—life, ability to earn a living, and general well-being of the patient—depends upon correct treatment. Practically, here, more than anywhere else, the principal problem for the diagnostician is that among the marvellous variety of the phenomena of disease he recognize with a clear vision those which offer a favorable prospect for treatment. This demand is at the present day still often unsatisfactorily fulfilled.

PRELIMINARY REMARKS ON ANATOMY AND NORMAL AND PATHOLOGICAL PHYSIOLOGY.

Nothing more than a sketch of what is most important can be given here. For more exact studies I refer to Edinger's well-known excellent work.[1]

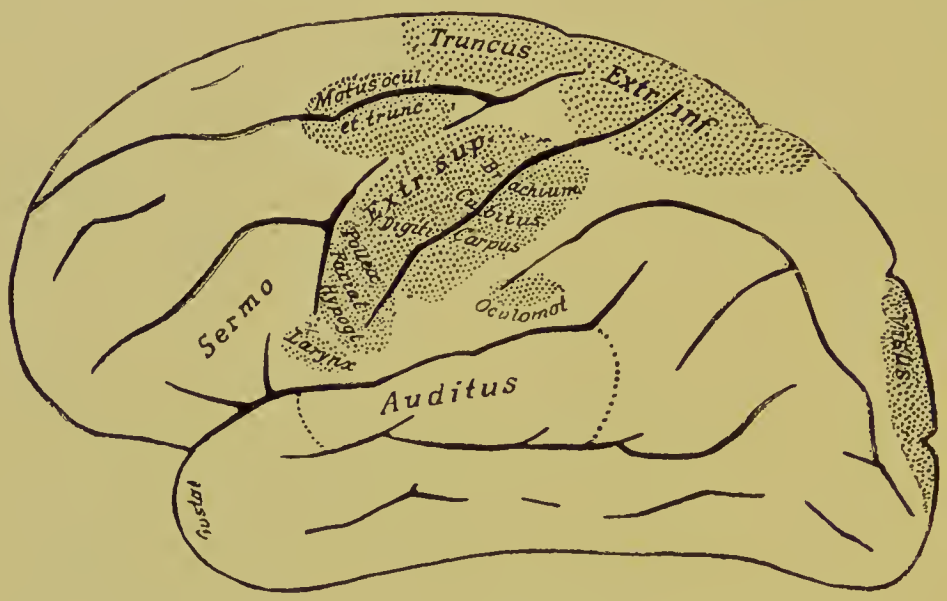


FIG. 155.—Localization of the cortex of the left hemisphere (after Edinger).

Without dwelling upon the general anatomy, we enter at once upon those parts of the nervous system which are specially concerned in diagnosis.

1. The Motor Tracts and Centers.

The motor tract for all regions of the body is divided into a *central* and *peripheral* one.

The *central motor tract, the pyramidal tract*, originates in the motor (the psychomotor) centers of the cortex of the cerebrum. These latter unite in the upper two-thirds of the so-called motor-cortex field, which includes the upper central convolutions and the lobus paracentralis of both hemispheres.

Fig. 154 shows the surface of the left hemisphere, with the names of the convolutions and the sulci.

From Fig. 155 may be seen where the muscular system of the face (tongue and eyes), of the larynx, of the trunk, and of the extremities has its origin within the motor zone.

And now to bring out the most important features: The center for the larynx, the hypoglossus, and the lower facial is situated in the lowest portion of the *anterior central convolution*, near the speech-center (the center of the anterior facial has not yet been positively made out); close to this, mostly in the *anterior central convolution*, is located the arm-center, and in such a way that its lower part presides over the distal part of the extremity; in the upper part of the *anterior* and *pos-*

[1] *Bau der Nervösen Centralorgane*, 5 Auflage, Leipzig, F. C. W. Vogel.

terior central convolution and are the *lobus paracentralis* is situated the center for the leg.

It will be noticed that the centers are tolerably wide apart, as is also the case with the tracts which originate from them and radiate into the medullary layer. Farther down the tracts converge in the *corona radiata*, in a fan-shape, to the internal capsule, where they lie close together in its posterior peduncle—that is, between the lenticular nucleus and optic thalamus: they lie close together behind a point midway between these [but do not connect with them]. Farther on they reach to the foot of the crus cerebri, and pass about in the middle of it. In the pons the pyramidal tracts are split up by transverse fibers. They unite again to form pyramids at the *anterior portion of the medulla oblongata*, and here the pyramidal tracts of both sides of the middle line lie very closely together. [From the circumstance that they form the anterior pyramids of the medulla they receive their name, "pyramidal tracts."] In the lower decussation of the pyramids of the medulla oblongata the right and left pyramidal tracts interlace, so that very much the larger part of the fibers go to form the *lateral column of the opposite side of the spinal cord* (*lateral pyramidal tract*, *Py-L*). Only a small part of the fibres [of the external aspect of the pyramids], without crossing to the opposite side, pass to the *anterior column of the spinal cord* [forming the columns of Türck]. (*Anterior pyramidal tracts*, *Py-V.*)

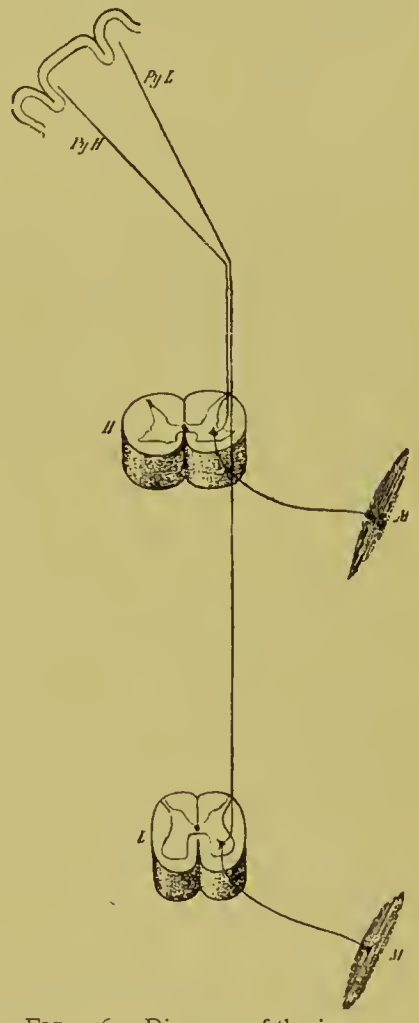


FIG. 156.—Diagram of the innervation of the muscles (partly from Edinger).

The radiation of the *Py*-tracts varies at different portions of the cortex (see text); *Py-H*, pyramidal tract for the cervical spinal cord; *Py-L*, pyramidal tract for the lumbar portion of the cord; *H*, cervical cord; *L*, lumbar cord; *Py-V* is omitted. Notice that down to the lumbar portion of the cord *Py-L* passes in the lateral column.

At different levels of the cord, from the lateral pyramidal tracts, successive fibers continually connect with the *ganglion-cell groups* of the anterior horn of the same side, and from these ganglion-cells arise the anterior roots of the [nerves of the] spinal cord. These unite with the posterior and form with them the mixed peripheral nerves. In these the motor tracts pass to the muscles.

The path of the *motor cerebral nerves* from the cortex to the nuclei of the pons and oblongata is not known, except that of the lower facial and the hypoglossus. The fibers for the former[1] pass from the cortical center obliquely across the lenticular nucleus to the internal capsule, where they lie close to the pyramidal tract. They pass with the latter

[1] Compare Fig. 157.

through the foot of the cerebral peduncle, but separate from it above the pons to reach the decussated facial nucleus in the lower part of the pons (Edinger). The *hypoglossus tract* runs near the speech-tract, likewise to the inner capsule, where it probably comes to lie between the fibers of the facialis and those of the extremities. In the pons it

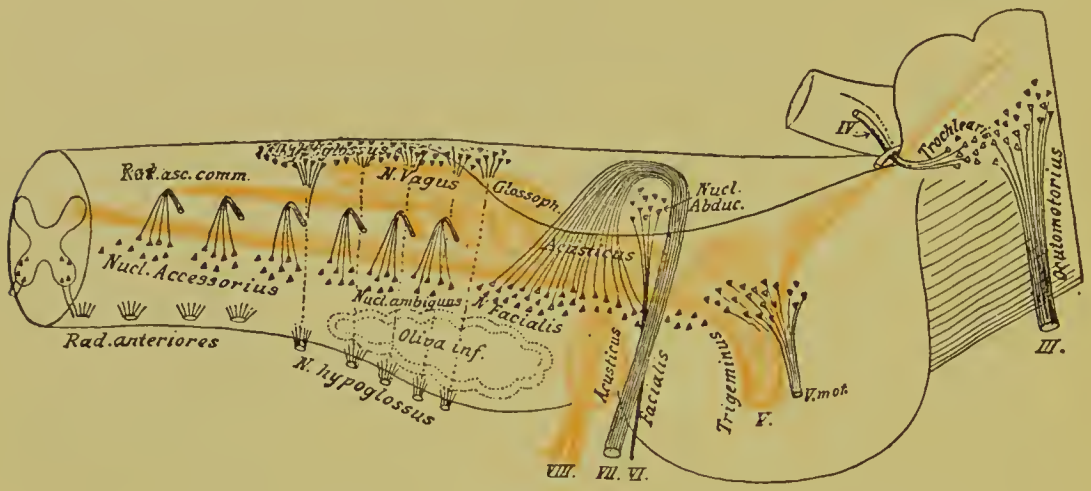


FIG. 157.—Location of the nuclei of the cranial nerves (Edinger).

The oblongata and pons are represented as transparent. The nuclei of sensation are red, those of motion are black.

separates from the pyramidal tract, and in the oblongata goes to the twelfth nucleus of the other side.

The tract for the *co-ordination of speech* has been determined for only a certain portion of its length. It runs from the third frontal convolution ("sermo" in Fig. 155), below the island [of Reil], almost horizontally to the internal capsule near its knee, and from there to the foot of the crus cerebri, to separate in the pons from the pyramidal tract and to end in the seventh and twelfth nuclei.

The *peripheral motor tract* is the designation given to the portion from the ganglion-cells of the anterior-horn ganglia or from the cells of the gray nuclei of the pons and oblongata through the motor cerebral and spinal nerves to the muscle end-plates. The nuclei and tracts of the cerebral and spinal nerves are known exactly throughout their courses. Fig. 157 illustrates the location of the nuclei in the pons and oblongata, represented as transparent. This figure, taken from Edinger's work, is admirably adapted for locating diagnostically these centers when they are the seat of disease.

The *cerebral nerves* in part have a complicated course among the peripheral nerves. In the first place, it is essential to observe that they may enter into local relation with different parts of the brain, not only at the place of their egress from the brain, but also in their course at the base of the brain, and may therefore participate in diseases, particularly tumors, at the base. This is the case with the facialis after its exit on the floor of the fourth ventricle, close to the lower surface of the cerebellum, and with the hypoglossus, which runs laterally upward beside the oblongata. The trigeminus penetrates the basal lateral part of the pons; the oculo-motorius penetrates the cerebral peduncle

and lies close to its base for a considerable distance more, etc. Moreover, it is of diagnostic interest to observe how the cerebral nerves at the base enter into relations among themselves by reason of their juxtaposition. Upon this point Fig. 159 gives satisfactory illustration. Finally, there also comes into consideration the manner in which the nerves enter the bony canals at the base of the skull, and partly also outside of it.

The *centers of the cortex* are those of voluntary motion. They transmit the stimulus through the fibers of the corona radiata or of the pyramidal tract respectively, to the nuclei of the oblongata or the anterior columns respectively, and these in turn transmit the irritation on through the peripheral nerves to the muscles. But likewise the nuclei of the peripheral motor tract are simultaneously *reflex centers;* that is, they transpose sensible stimuli transmitted from the periphery into motor stimuli for the muscles belonging to them, and they are also trophic centers for the peripheral motor nerves and the muscles; that is, they preside over their nutrition.

The cutting out of the cortical center or the interruption of the central motor tract belonging to them renders voluntary movements in the respective muscular region impossible—a paralysis of voluntary movements. At the same time, the respective muscles remain under the influence of reflex stimuli, and their nutrition also for the most part remains normal, since the nuclei pertaining to the peripheral motor tract continue to perform their function. On the contrary, the reflexes and tonus of the muscles usually are increased, and hence these paralyses in general are called *spastic paralyses.*

If, however, the *nuclei of the peripheral tract* are destroyed or the fibers going from them are interrupted, the appertaining muscles are completely paralyzed; that is, they are excitable neither through the will nor through the reflexes, they immediately lose their tonus, and they degenerate, because their trophic stimulus is removed. Only mechanical and electric—that is, galvanic—stimuli, which directly act upon the muscular fibers, produce unnaturally slow contractions. These paralyses (*nuclear and peripheral paralyses*) are therefore correctly called "*atonic degenerative paralyses.*"

Both of these kinds of paralysis are of great significance for diagnosis, because we are able immediately to distinguish whether a lesion has affected a central or a peripheral motor tract. For the more exact determination of the seat of disease it is necessary in the first place to ascertain the location of the paralysis and its possible combination with disturbances of the sensible sphere, with other signs on the skull, spinal column, extremities, etc.

2. The Sensitive or Centripetal Tracts.

The tract of the voluntary and involuntary sensibility of the skin of the trunk and of the extremities passes from the sensitive terminal fibers of the skin in the mixed nerves, then into the posterior root to the cord. Until a very short time ago very little was known of its continuation upward. More recent investigations, however, especially Edinger's excellent works, have shown that it gives two (possibly more) processes

to the posterior root—one which ascends without decussating in the posterior columns to the medulla oblongata, and there, by interlacing fibers from ganglia (posterior column ganglia), enters the decussated loop [or crossed fillet] (lemniscus), and thence into the deep medulla of the corpora quadrigemina. A second process enters the spinal-cord ganglia—namely, those belonging to the gray posterior horns, the *end-nuclei of the peripheral sensible tract.* From these end-nuclei arises *the central sensitive tract,* which partly crosses directly and partly higher in the spinal cord through the anterior commissure, and reaches the anterior and lateral column on the opposite side as the tractus tecto-spinalis, to pass to the loop (lemniscus), and to pass with this to the corpora quadrigemina. For a still farther distance we know the course of the sensible tract in the brain, for we know that it enters through the tegmentum of the cerebral peduncle into the inner capsule, behind the pyramids; that is, into the posterior third of its posterior peduncle. We have no positive knowledge of its distribution upward beyond this point.

The tract of *deep sensibility* (usually called the muscular sense) seems to run in the posterior columns. Most probably it ends in the *motor cortical zone* of the central convolutions and the lobus paracentralis.

Moreover, *the lateral column of the tract of the cerebellum* is centripetal, which, arising from the columns of Clarke, in the upper portion of the cord, goes into the cerebrum. Its function also is not entirely clear; probably it is of service in preserving equilibrium.

Severe lesions, or complete interruption of the tract of sensibility of the skin in the peripheral nerves or in the cord or in the internal capsule, causes *total anesthesia* of the skin. If the lesion is not severe, there is diminution of the sense of touch or a partial loss of sensibility —a *partial paralysis of sensibility*, as the sense of pain, of cold, of heat; and this latter is frequent, especially in diseases of the spinal cord. Anesthesia from local disease of the internal capsule or of the spinal cord manifests itself upon the opposite side.

3. Centers and Tracts of the Special Senses.

(*a*) **Sight.**—This tract passes from the retina in the eye to the chiasm. Here occurs a peculiar partial decussation (semi-decussation), which is reproduced in Fig. 158: the optic-nerve fibers belonging to the outer half of the retina do not cross, those belonging to the inner half do. Then it passes in the optic tract to the anterior corpus quadrigeminum, and from there in the posterior third of the posterior limb of the internal capsule entering into relation with the pulvinar of the optic thalamus and the corpus geniculatum ext., and then spreads out obliquely backward and upward in the cortex of the occipital lobe. The most important points in relation to this nerve are the following:

1. That pathological processes at the base of the brain, and also lesions in the posterior end of the inner capsule (causing a simultaneous hemianesthesia), lastly, of the pulvinar of the optic thalamus, or of the occipital lobe, produce disturbances of vision.

2. That every complete destruction of the cortical centers in the occipital lobes, as well as of the tract from there to the chiasm, cuts off the impressions of sight from the outer half of the retina of the same side and the inner half of the opposite, thus from synonymous halves of the two retinæ. Thus, hemiopia and hemianopsia are produced.[1]

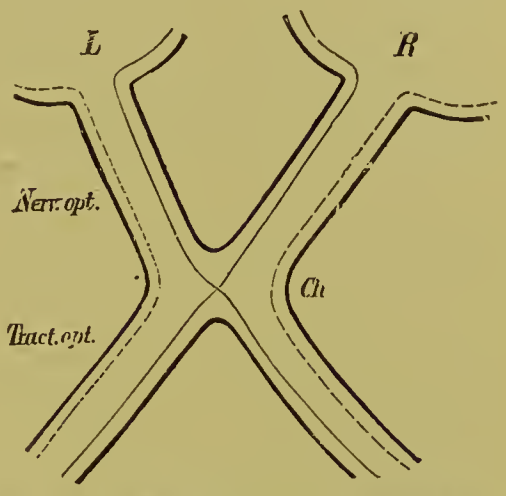


FIG. 158.—Diagram of the optic-nerve fibers in the chiasm.

(*b*) **Hearing.**—The acoustic nerve, together with the facial, passes to the oblongata, etc., to the acoustic ganglion, in regard to which we cannot here enter into further detail. In its central course the auditory tract lies in the tegumentum of the opposite cerebral peduncle, and then appears probably in the most posterior sensitive portion of the internal capsule, whence it radiates in the cortex of the temporal lobe.[2] It is also connected with the cerebellum.

(*c*) **Smell.**—Of the olfactory nerve perhaps nothing more is to be said than that its centripetal tract seems to pass through the posterior portion of the internal capsule.

(*d*) **Taste.**—The sense of taste is located [chiefly] in the glosso-pharyngeus nerve, distributed to the palate and the posterior third of the tongue, by which nerve it is conveyed to the oblongata. The course for the anterior two-thirds, however, is complicated: as the chorda tympani it first passes in the lingual nerve, but leaves this and goes to the facial, leaves this again at the geniculate ganglion, and probably extends, as the greater superficial petrosal nerve, Vidian, and the spheno-palatine ganglion, to the second branch of the trigeminus, going with this toward the center, or through the otic ganglion to the third branch of the fifth nerve (Ziehl). We again meet the fibers of taste in the posterior portion of the inner capsule.

It is very important in peripheral paralysis of the facial to note the participation of the sense of taste at the anterior portion of the tongue, and also (according to Erb and others) in disease of the trigeminus situated near its origin, as well as in lesions of the posterior portion of the inner capsule (hemianesthesia).

Until we come to the Symptomatology we delay speaking of all other points regarding localization of the brain, especially regarding aphasia and the phenomena associated with it, and regarding the origin of certain forms of convulsions, of vertigo, co-ordination, etc.

4. Remarks upon the Vessels Supplying the Brain.

The brain is supplied with blood from the two internal carotids and from the vertebral artery. The right and left vertebrals unite at the basilar surface of the pons to form the basilar artery; this, again,

[1] See under Eye.

[2] See Word-deafness.

divides at a point corresponding to the anterior inferior border of the pons into the two posterior cerebral arteries, which, by the posterior communicating arteries, form a connection with the carotids (the circle of Willis). Besides the ophthalmic and the posterior communicating, the carotid gives off the anterior communicating, which, with its opposite fellow, completes the circle of Willis. There also arises from the carotid the middle cerebral, the [largest and] most important vessel of the brain.

Of these vessels the greatest interest attaches to those which supply the pons and medulla and the most important part of the cortex and the internal capsule.

The pons and medulla are chiefly supplied by the basilar and vertebrals. The branches of these are terminal arteries; that is, they do not anastomose with each other or with other branches in their neighborhood. Hence, thrombosis or emboli of such branches—or, for instance, of a part of the basilar—immediately produces arrest of function, and if this arrest continues for a time—that is, unless the stoppage is soon removed—there follows anemic necrosis of the affected portion of the pons or medulla.

The region of next importance is that supplied by the middle cerebral artery (the artery of the fossa of Sylvius). This, as well as the regions of the cerebrum supplied by each of the two other arteries supplying portions of the cerebrum, divides distinctly into two parts, which do not anastomose with each other into an inner and a cortical portion. *The inner region*, supplied by the middle cerebral artery and its branches, embraces the internal capsule, with the exception of its posterior section (sensory tract), the lenticular nucleus, the greater part of the caudate nucleus, and a part of the optic thalamus. This internal region of the middle cerebral artery (artery of the fossa of Sylvius) is sharply distinguished from the neighboring regions of the other arteries of the brain: there are no anastomoses; hence, continuous occlusion of this vessel at its root must inevitably result in softening of the above-named central portion of the brain. The cortical region of the middle cerebral artery extends over the third frontal convolution, the anterior central convolution (with the exception of the upper portion, which belongs to the anterior cerebral artery), the posterior central convolution, the superior and inferior parietal lobes, the whole region in the neighborhood of the fissure of Sylvius; lastly, the second and third temporal convolutions. This cortical region of the artery of the fossa of Sylvius seems to anastomose in a very distinctive way with the neighboring cortical regions; for this reason occlusion of the middle cerebral artery in only a part of the cases results in softening of this cortical portion of the brain.

The optic center of the occipital lobe, the corpora quadrigemina, and the posterior portion of the internal capsule are supplied by the posterior cerebral artery.

The predominant importance of the middle cerebral artery consists not only in the fact that it supplies the most important portion of the cerebrum, but also because it is within this region that both hemorrhages and emboli most frequently occur. These two disturbances chiefly affect the internal region of the artery—the hemorrhages probably because

the pressure is highest in the branches that go directly off from its root, or that here is felt most strongly the rapid changes in the power of the heart; but emboli much more frequently disturb the inner territory than the cortical, because, as was mentioned before, there are no anastomoses in the former region, while in the cortical there are. In the relation of the left carotid to the aorta (going off at a very acute angle) seems to lie the explanation as to why emboli are much more frequent in the left middle cerebral artery than in the right.

5. Topographical Diagnosis of Diseases of the Brain and Spinal Cord.

Local diseases of the brain, whether hemorrhages, softening, local inflammation, cysts, abscesses, or tumors, cause symptoms which may in general be divided into two classes: one class form the *local symptoms*—that is, those which occur in such a way that the local disease functionally damages a certain definite circumscribed portion of the brain; and, secondly, the *general phenomena* which proceed from a more or less uniform injury of the whole brain, especially by concussion and by increased intracranial pressure.

The local symptoms are divided into direct and indirect, or local symptoms and neighboring symptoms (Edinger). The *direct local symptoms* arise from destruction of functionally important portions of the brain or of parts whose functions cannot be performed by others. Indirect local symptoms are caused by temporary injuries or those which may be recovered from. The injuries often change: the damage which the real focus produces falls upon the tissue immediately about it, but often also at a great distance from it. These injuries, passing beyond the original focus of injury, result from commotion produced by cerebral hemorrhages, from congestive states, in tumors of the brain, which cause the tumor to enlarge and thus give rise to pressure-anemia in the neighborhood; likewise by hyperemia, hemorrhage, collateral edema, etc., according to the nature of the local disease.

The direct local symptoms are always persistent unless, after a long space of time, a compensation of the omitted functions is made up by practice. Irritation phenomena—as, for instance, dissociated spasms of neuralgia—are, strictly speaking, never direct local symptoms, for they never occur other than only temporarily.

Indirect local symptoms are partly phenomena of irritation and partly of defective function, and they may disappear altogether or only partly. From such changing symptoms not infrequently permanent ones may develop from the neighborhood of the focus of injury, and in this way they come into the class of direct local symptoms. This latter phenomenon occurs especially in tumors.

General phenomena are sudden shock-like conditions, as in apoplexy or slowly developing symptoms of pressure. They consist of disturbances of consciousness, attacks of dizziness [vertigo], unlocalized headache, obtusion of the sensorium, of amnesia with respect to answering questions or slowness of speech; in short, general dulness ("stupor"), sometimes also a sort of marasmus; general epileptiform spasms; vomiting; retardation of pulse; lastly, the extremely important symp-

tom of choked optic disk, resulting in subsequent atrophy of the optic nerve.

In *local diseases of the spinal cord* the general phenomena usually do not play an important rôle, and we are not able sharply to separate direct from indirect local symptoms.

The following facts are essentially determinative in judging of the local symptoms:

1. If the lesion is located in the cortex in the central motor tract

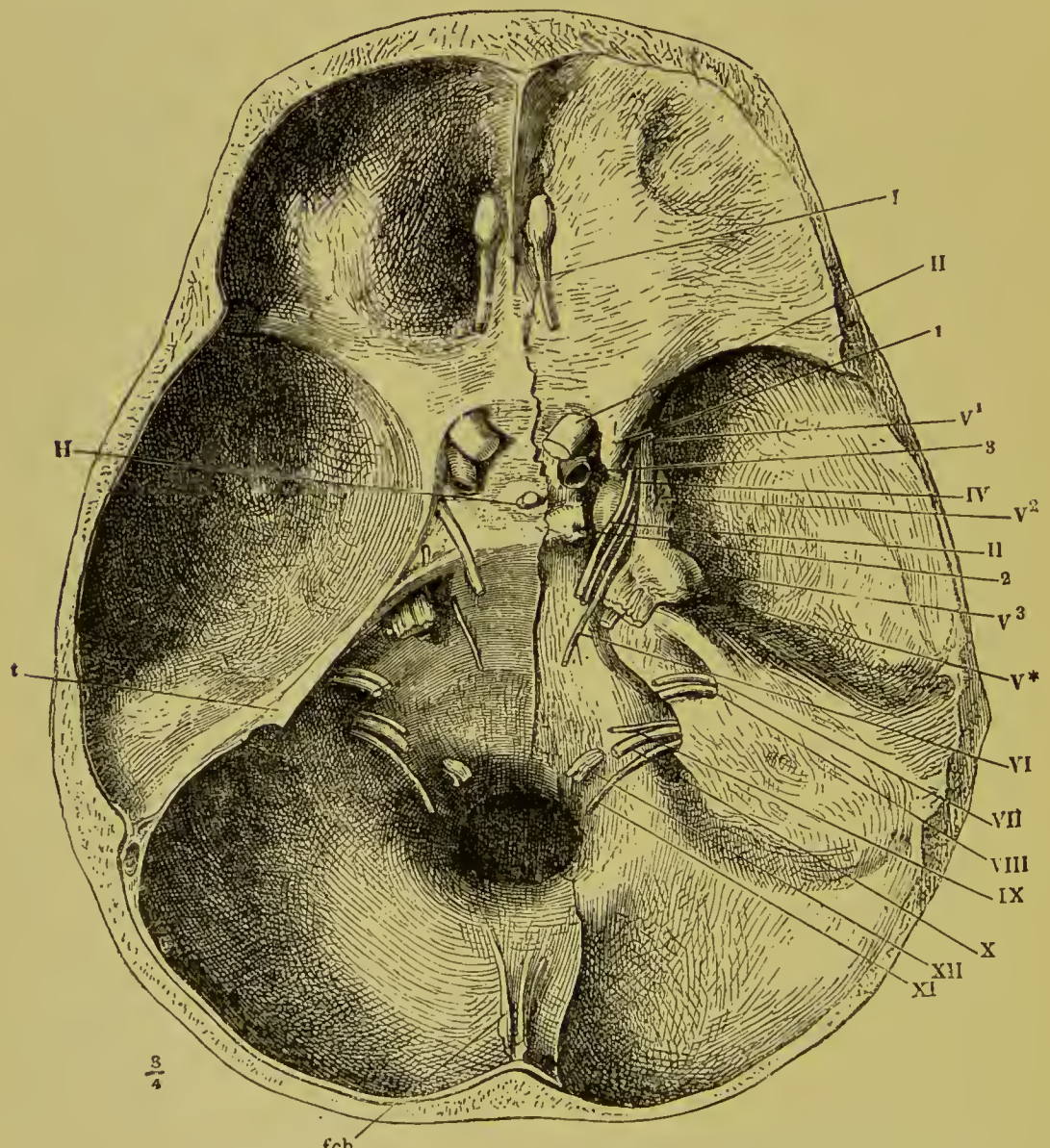


FIG. 159.—Points of exit of the cranial nerves from the skull (Henle).

The Roman figures indicate the cranial nerves; V^1, V^2, V^3, first, second, and third branches of the trigeminus; V^*, Gasserian ganglion.

above the point of decussation, then the paralysis is upon the opposite side of the body; lesion of a tract below its decussation produces paralysis of the same side. It is to be pointed out that the central motor tract for the trunk and extremities decussates rather closely together

in the decussation pyramid, while the tracts of the motor cerebral nerves decussate higher up, the facial tract about in the middle of the pons, and the hypoglossus tract in the oblongata. Hence the decussated facial tract is in the lower part of the pons, lying close to the still undecussated tract of the extremity of the opposite side. A lesion, therefore, which affects the lower part of one-half of the pons may produce paralysis of the opposite side of the body and of the same side of the face—*hemiplegia cruciata seu alternans*, an important symptom of disease of the pons (compare Fig. 160).

2. If the lesion affects a cortical center or a point in the pyramidal tract in the brain, the pons, oblongata, the spinal cord above the point of entrance of the particular tract into ganglia of the anterior horn (or analogous gray nuclei of the oblongata or of the pons), then, because the trophical influence of the cortical center from above ceases at that point, the affected tract degenerates just up to the corresponding cells of the anterior horn, while these and the peripheral nerves and the muscles do not degenerate. This degeneration of the pyramidal tract does not in itself cause any further clinical phenomena. On the other hand, if the lesion is in the anterior horn or downward from there in the motor tract, there is degeneration downward of the nerves and of the muscles supplied by the portion which is the seat of the lesion; and this may be recognized clinically from the signs of degenerative atrophy (*relaxed paralysis, diminution in volume of muscle, reaction of degeneration in electrical examination*).

3. Since the centers and tracts in the different sections in some instances lie wide apart and in others close together, a given extent of lesion, according to its location, will cause a paralysis widely different in its area:

(*a*) A lesion of considerable extent located in the cortex, or in the corona radiata just under it, generally affects only the center for one-half of the countenance, or an arm, or a leg (*monoplegia, dissociated paralysis*). There is also another important symptom which is characteristic of irritation of the cortex: spasms *precede* paralysis, and also linger after the paralysis disappears, in the area supplied by the affected portion of the cortex—"*dissociated spasms*." These spasms may extend to other parts of the body, but the part corresponding to the affected portion of the cortex is always the "primary seat of spasm."

(*b*) If located in the internal capsule, then the lesion need not be so very large in order to produce *paralysis of the whole of the opposite side of the body—hemiplegia*. This points to the crus cerebri and also to the pons.

(*c*) If the lesion is in the pons and oblongata, even though of slight extent, it affects fibers of the central tract as well as nuclei of the cerebral nerves; it readily causes injury to centers that are very essential to life, *the respiratory center, vagus center for the heart*, and death may soon follow; if there is hemorrhage or softening, it often takes place immediately.

4. It is of considerable diagnostic importance that at certain places some of the cranial nerves and the pyramidal tracts for the extremities lie in juxtaposition, and also that at the base of the crus cerebri the oculo-motorius passes near the pyramidal tract for the arm and leg of

the opposite side; at the lower part of the pons the pyramidal tract again runs near the tract of the facial after its decussation (compare Fig. 160).

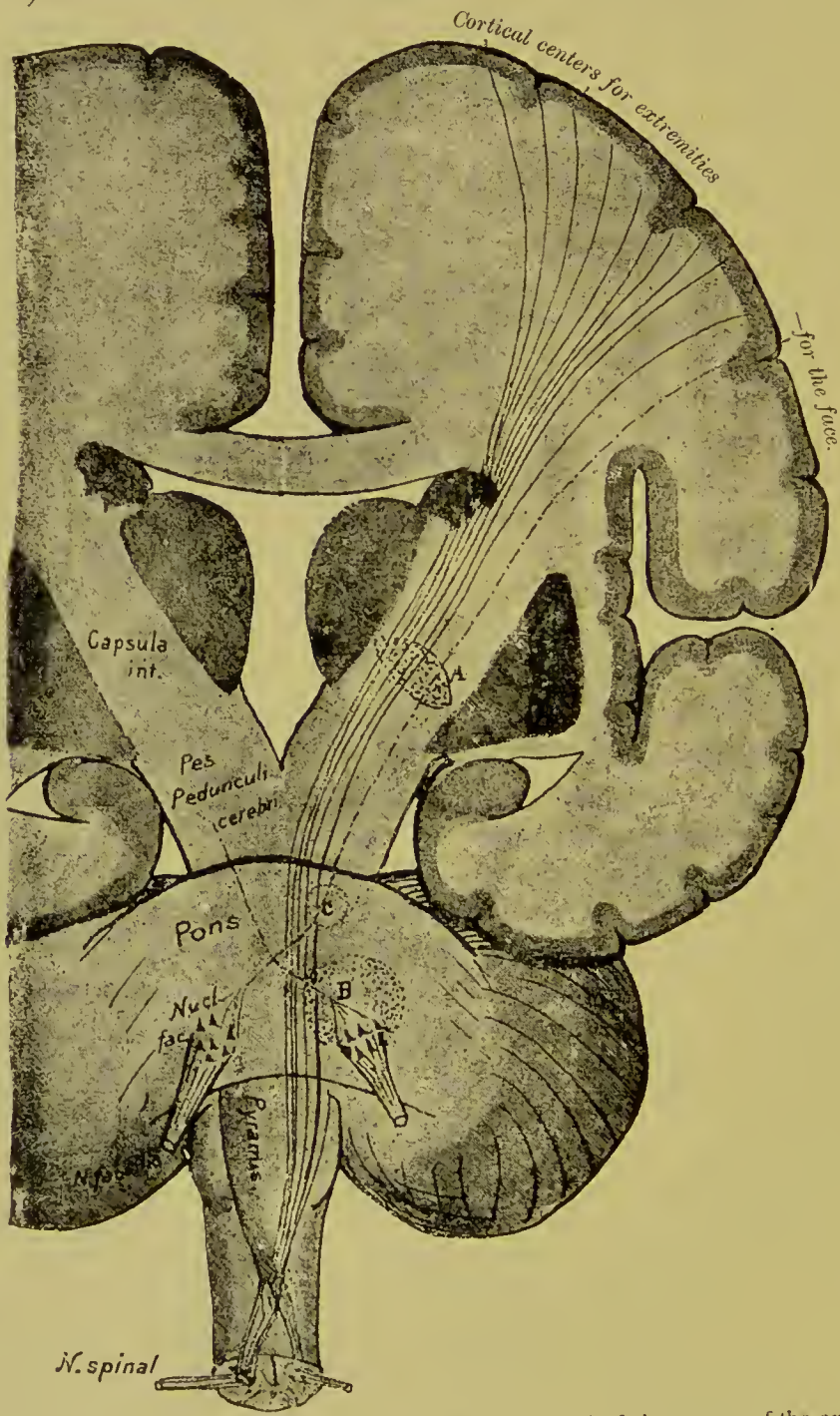


FIG. 160.—Diagram of the motor tracts of the facial nerve and of the nerves of the extremities (Edinger).

At *A*, *B*, *C*, are indicated supposed local diseases. *A*, lesion of the left side of the internal capsule, causing right hemiplegia on the right side; *B*, lesion of the left half of the pons, touches the pyramidal tract of the extremities of the right side and of the left facial, causing crossed paralyses: *C*, shows the rare condition of uncrossed facial paralysis and paralysis of the extremities from lesion in the pons.

A tumor at the base of the crus cerebri can therefore cause paralysis of the oculomotorius of the same side and of the arm and leg of the

opposite side ("crossed paralysis of the motor oculi and extremities"—"*Weber's syndrome*"); on the other hand, a lesion in the lower portion of the pons, as has already been explained, may cause paralysis of the facial of the same side and of the extremities of the opposite side ("crossed paralysis of the facial and extremities").

If the lesion is higher in the pons, above the decussation of the facial, it may cause paralysis of the facial and extremities of the opposite side; that is, a true hemiplegia (which is very rare) (compare Fig. 160).

It has recently been discovered that paralysis of the oculo-motorius of one eye and tremor or disturbed co-ordination of the opposite arm point to the peduncle of the cerebellum (*Benedict's symptom-complex*).

5. *A tumor or an inflammatory affection at the base of the brain*, according to its precise seat, injures the cerebral nerves passing there. If the focus is in the anterior cranial fossa, the olfactory is implicated; if located in the middle cranial fossa, the opticus, the oculo-motorius, trochlearis, abducens, and sometimes also the olfactory, may become diseased. If in the posterior cranial fossa, then the trochlearis, abducens, facial, acusticus, glosso-pharyngeus, vagus, accessorius, and hypoglossus come into consideration.[1] The disease may be bilateral. As has been partly explained above under 3, simultaneous injury of the cerebral peduncle, of the pons, and of the oblongata may implicate the pyramidal tracts: there is *paralysis of the extremities.*

In *diseases of the optic thalamus* there have been observed homonymous hemianopsia, hemichorea, hemiathetosis, and one-sided tremor; but it is uncertain whether a part of these symptoms are not caused by the internal capsule, which lies close by. Foci in the corpus striatum cause the same difficulty.

Foci in the cerebral peduncle, besides motor paralysis, not infrequently cause sensible and vaso-motor paralysis of the opposite side. Crossed paralysis of the extremities and the oculo-motorius, moreover, may be caused not only by a tumor at the base of the cerebral peduncle, but also by a focus under the corpora quadrigemina; in both cases paralysis of the third nerve is often complete—ptosis, dilatation of the pupil, and outward rotation of the eye by the adducens.

If only one hemisphere participates in the disease, affections of the cerebellum often produce only indefinite or general symptoms. Phenomena of extreme diagnostic importance arise from the participation of the vermiform process, and by neighborhood symptoms from the corpora quadrigemina, pons, and oblongata or their nerves. Lesion of the vermiform process causes cerebellar ataxia, vertigo, and "riding-school gait" [cerebellar gait]; the neighborhood symptoms are paralysis of the third and fourth nerves (corpora quadrigemina), and paralysis of the seventh nerve from the pons as well as from the floor of the fourth ventricle, paralysis of the sixth nerve, and trigeminal neuralgia. Among the general phenomena to be mentioned are the following: relatively early and well-marked choked optic disk, occipital headache, vomiting without ascertainable cause, convulsions, and compulsory position upon the side.

Localization of Disease in the Spinal Cord. — In the

[1] Also see Fig. 159, which shows how the individual cerebral nerves run together at the base.

first place, particular significance attaches to the cervical and lumbar enlargements of the cord and the accumulation of nuclei in the anterior horns for peripheral innervation of the extremities. It has been ascertained that within the enlargements lies the localization for individual parts of the muscle-system of the extremities, but our knowledge of the functions of the motor nuclei in the different sections of the dorsal cord is less exact. Much has also been learned, by comparison of cases of disease, regarding the representation [mediation by nuclei] of conscious sensibility of the skin and of deep sensibility [sensibility in the deeper parts], which produce reflexes in the spinal cord.

The simplest way to determine the seat of lesion in the different segments of the spinal cord is to keep in mind the height of entrance of the anterior posterior roots. These are given in Starr's table, which, with the author's permission, we copy from Edinger's work, already referred to several times. The table also contains the localization of the reflexes. It is well to read what has been said above about reflexes in order to understand this table.

Localization of the Function of Different Segments of the Spinal Cord.

Segments.	Muscles.	Reflexes.	Innervation of sensation of skin.
Second and third cervical.	Sterno-mastoid. Trapezius. Scaleni and muscles of the neck. Diaphragm.	Inspiration on sudden pressure under the arch of the ribs.	Neck and occiput.
Fourth cervical.	Diaphragm. Supra- and infra-spinatus. Deltoid. Biceps and coraco-brachialis. Supinator longus. Rhomboidei.	Dilatation of pupils after irritation of the neck, fourth to seventh cervical.	Neck, upper region of shoulder, outer side of the arm.
Fifth cervical.	Deltoid.	Scapular reflex, fifth cervical to first dorsal.	Posterior portion of shoulder and arm.
	Biceps and coraco-brachialis.	Tendon reflexes of the respective muscles.	Outer side of the upper and the forearm.
	Supinator longus et brevis. Pectoralis—clavicular portion. Serratus magnus. Rhomboidei. Brachialis anticus. Teres minor.		
Sixth cervical.	Biceps. Brachialis anticus. Pectoralis—clavicular portion. Serratus magnus. Triceps.	Reflexes from tendons of the extensors of the upper and the forearm.	Outer side of the forearm.
	Extensors of the hand and fingers.	Tendons of the wrist, sixth to eighth cervical.	Back of hand, region of radialis.
	Pronators.		

Segments.	Muscles.	Reflexes.	Innervation of sensation of skin.
Seventh cervical.	Long head of triceps.	Blow upon the palm causes fingers to close.	
	Extensors of hand and fingers.		
	Flexors of hand.		Radial region of hand.
	Pronators of hand.		
	Pectoralis—costal portion.		
	Subscapularis.	Palmar reflex, seventh cervical to first dorsal.	
	Latissimus dorsi.		Distribution of median.
Eighth cervical.	Teres major.	Pupil reflexes.	
	Flexors of hand and fingers.		
	Small muscles of the hand.		
First dorsal.	Extensors of the thumb.		Ulnar region.
	Small muscles of the hand.		
	Ball of thumb and little finger.		
Second to twelfth dorsal.	Muscles of back and abdomen.	Epigastrium, fourth to seventh dorsal.	Skin of the chest, back, abdomen, and upper gluteal region.
	Erectores spinæ.	Abdomen, seventh to eleventh dorsal.	
First lumbar.	Ileo-psoas.	Cremaster reflexes, first to third lumbar.	Skin of region of pubes. Anterior side of scrotum.
	Sartorius.		
	Abdominal muscles.		
Second lumbar.	Ileo-psoas.	Tendon of patella, second to fourth lumbar.	Outer side of hip.
	Flexors of knee (Remak?).		
	Quadriceps femoris.		
Third lumbar.	Quadriceps femoris.		Anterior and inside of thigh.
	In-rotators of the thigh.		
	Adductores femoris.		
Fourth lumbar.	Adductores femoris.		Inner side of hip and leg to the ankle.
	Adductores femoris.		
	Tibialis anticus.	Gluteal reflexes, fourth to fifth lumbar.	Inner side of the foot.
	Flexors of the knee (Ferrier?).		
Fifth lumbar.	Out-rotators of the hip.		Back of hip, upper part of thigh, and outer part of the foot.
	Flexors of knee (Ferrier?).		
	Flexors of foot.		
	Extensors of toes.		
	Peronei.		
First and second sacrals.	Flexors of foot and toes.	Plantar reflexes.	Back of upper part of thigh, outer side of leg and foot.
	Peronei.		
	Small muscles of foot.		
Third to fifth sacral.	Muscles of the perineum.	Tendo Achillis.	Skin over sacrum, anus, perineum, genitals.
		Bladder and rectal centers.	

METHOD OF EXAMINATION.

Examination of the Seat of Disease.

From the physiological properties of the nervous system it follows, from what has hitherto been said, that when affected by disease there is little or nothing to be seen at the seat of the disease, while the symptoms are manifest at other portions of the body often quite distant from it. Besides, the brain and spinal cord are almost entirely removed from the possibility of being examined on account of their bony casements. Lastly, very often a local disease of the nervous system, although it causes pronounced phenomena, is locally very indistinct. For all these reasons the local examination of the nervous system in a number of its diseases is quite subordinate. Still, we place its consideration first because in a systematic examination it belongs there, and because the expression of our opinion cannot at all affect the value which it nevertheless in many respects possesses.

The Skull.—The majority of the diseases of the brain and its coverings run their course without any manifest effect upon the skull; indeed, there is no disease of that organ in which it may not more or less frequently happen that alterations in the skull were entirely wanting. If there are such alterations in a portion of the cases they are secondary in their nature, dependent upon disease on the inner surface; in other, more rare, cases the alterations of the skull are the cause of the disease of the brain.

As methods of examination we mention *inspection*, *palpation*, and *measuring* or tracing the shape of the cranium upon paper.

The Size of the Cranium.—Generally this is determined by the circumference of the head over the glabella and the occipital protuberance, and by estimating the relation between the brain-case proper and the face. This latter can be measured simply by the eye. In the newly-born the circumference of the head is 39 to 40 cm. (according to others somewhat less). In the course of the first year it increases to about 45 cm., and from then to the beginning of the twelfth year to 50 cm.; in adults it amounts to about 55 cm. (in women it is generally somewhat less than in men.

Marked enlargement of the cranium, *macrocephalus* (to 80 cm. and more in circumference), occurs with *hydrocephalus* if the fontanelles have not yet closed. Then the frontal bones particularly project; the countenance is proportionally too small, the eyes are directed downward, the expression is often peculiarly staring; the fontanelles are very large and remain open for a long time; the cranial bones are thin. Hydrocephalus which occurs later, when the skull has already closed, causes little or no enlargement of the head.

Moreover, a somewhat considerable *macrocephalus* is peculiar to the rachitic skull, and is here dependent upon thickening of the cranial bones. But it is generally somewhat angular (caput quadratum). There is no notable recession of the bones of the face as in the former; the bones give the impression of being dense, only the occipital bone is sometimes very thin, even as paper, sometimes upon pressure crackling like parchment (be careful!). Here, too, the fontanelles remain

open abnormally long—sometimes into the third year. The distinction from hydrocephalus is made in the first place by an examination of the nervous system, which in this disease is almost always injuriously affected (as respects its psychic, intellectual, and motor functions), while in rachitis it is normal; also the evidences of rachitis are to be sought at other points (the inferior maxilla, the thorax, the bones of the extremities). Moreover, we may have a combination of hydrocephalus and rachitic thickening of the cranium.

Abnormally small skull, *microcephalus*, is naturally connected with abnormally small brain, thus necessarily with idiocy.[1] The brain anomaly always seems to be the primary one.

Form of the Skull.—*Departures from the Typical Form.*—Here belong dolichocephalus, brachycephalus, and other forms of head which are often met with without any pathological condition of the brain, but also in congenital malformation of the brain, as in *idiots.* Asymmetry of the skull likewise occurs with this condition, but also not infrequently with persons who are perfectly healthy and intelligent. We discover the asymmetry of the skull by viewing it from above or by tracing it upon paper, measuring the sagittal and the large transverse diameters of the cranium with the calipers, and making an outline with a strip of lead, as was described upon page 140 in the examination of the form of the thorax.

Circumscribed projections and depressions have much greater pathological significance, the latter, however, very frequently not with reference to disease of the brain, but as signs of a general disease. *Projections* occur in disease of the cranial walls and of the dura mater,[2] and these are chiefly syphilitic gummata, carcinoma, and sarcoma. Sinkings-in, *depressions*, impressions, may be traumatic. If there is defect of the bony wall, the defect may feel like a fontanelle. Soft and slightly depressed [or depressible] round spots are sometimes present in carcinoma of the cranial vault. Very important, lastly, are scar-like, round depressions over which the scalp is adherent, and which often contain an actual scar: these occur as the result of healed syphilitic gummata or deep ulcerations. All these appearances, but especially the traumatic and syphilitic depressions, are of the greatest diagnostic importance. When the skull is thickly covered with hair they may be easily overlooked if we do not examine it with the greatest care by feeling all points.

The anatomical relations between the skull and the brain, especially the convolutions and fissures on the surface of the latter, have great diagnostic significance. When there is a circumscribed depression or a prominence from tumor it is important to determine what part of the cerebral surface is pathologically affected. Again, it often is important when there are local brain-symptoms, especially if they point to the region of the cortex, to examine with the utmost care the part of the skull which overlies that portion of the brain to which the symptoms point. Recently the latter aspect of this subject has become of greatest interest, because brain-surgery has advanced to such a point that

[1] See below.

[2] The knowledge and significance of tumors of the cranium caused by meningocele and cephalocele are taught in works upon surgery.

abscesses and circumscribed tumors can sometimes be operated upon. As these pathological conditions very often exist without a point of circumscribed tenderness upon the skull,[1] every direct sign of their location may be absent: their seat can only be determined indirectly from the immediate local symptoms.

Some data are here introduced for determining upon the skull the most important regions of the surface of the brain.[2] However, it must be borne in mind that the relation-space between the convolutions and furrows of the brain and the skull cap is not wholly constant, but rather varies within certain limits. For instance, the opening in the skull made by a trepan may miss the spot of the cortex sought for by as much as 5 cm. Hence, when in doubt, it is desirable to determine the location by applying a mild faradic current to the exposed cortex (Horsley), and then observe from which point the muscles of the arm, leg, or face of the opposite side are made to contract.[3]

Poirier, for convenience in determining locations, employs two lines, which he designates as the Rolando and Sylvian lines (see Fig. 161).

(*a*) The Rolando line is determined as follows: Mark with a blue pencil the upper angle of the zygomatic process of the temporal bone, then draw a line perpendicular to this process from just in front of the tragus—that is, between this and the posterior border of the temporo-maxillary joint. Upon this line, 7 cm. from the auditory meatus, is the lower end of the Rolando line. In the sagittal suture measure one-half of the distance from the apex of the naso-frontal angle to the occipital protuberance, plus 2 cm.: this gives the upper end or junction of the Rolando line with the sagittal suture.

(*b*) The Sylvian is a line drawn from the naso-frontal angle to a point 1 cm. above the lambda or 8 cm. above the external occipital protuberance.

The relation of these lines to the central fissure (the fissure of Rolando) and to the fissure of Sylvius is seen in the illustration. It also shows the local relation of the most important regions of the cortex to Poirier's lines. The center for the lower extremity corresponds to the upper third;[4] the center for the upper extremity, to the middle third; the center for sight and tongue, to the lower third of the Rolando line. The motor speech-center lies somewhat below and anterior to the latter. The temporal lobe is found between the Sylvian line and the external auditory meatus, while the center for understanding of speech lies just under the Sylvian line. Abscesses of the temporal lobe generally lie somewhat deeper, nearer the ear. The sight-center of the cortex corresponds with the posterior end of the Sylvian line, near the middle line.

When the external occipital protuberance is not distinct (adults), if the head is large, we may measure 18 cm., if small, 17 cm., from the naso-

[1] See below.

[2] In these we follow the statements of P. Poirier (*Topographie cranioencéphalique*, Paris, 1891), whose methods seem to us to be the best.

[3] For further information consult works upon Surgery.

[4] In trephining at this point the surgeon must keep 2 cm. from the middle line in order to avoid the longitudinal sinus. The lateral sinus lies in the prolongation of the line of the zygomatic process toward the external occipital protuberance.

frontal angle on the sagittal suture: this gives the upper end of the Rolando line.

In children the lower end of Rolando's line is found by measuring from the external auditory meatus to the sagittal suture: the point sought for lies about 15 cm. below the middle of this opening. According to Poirier, the other data do not differ essentially in children.

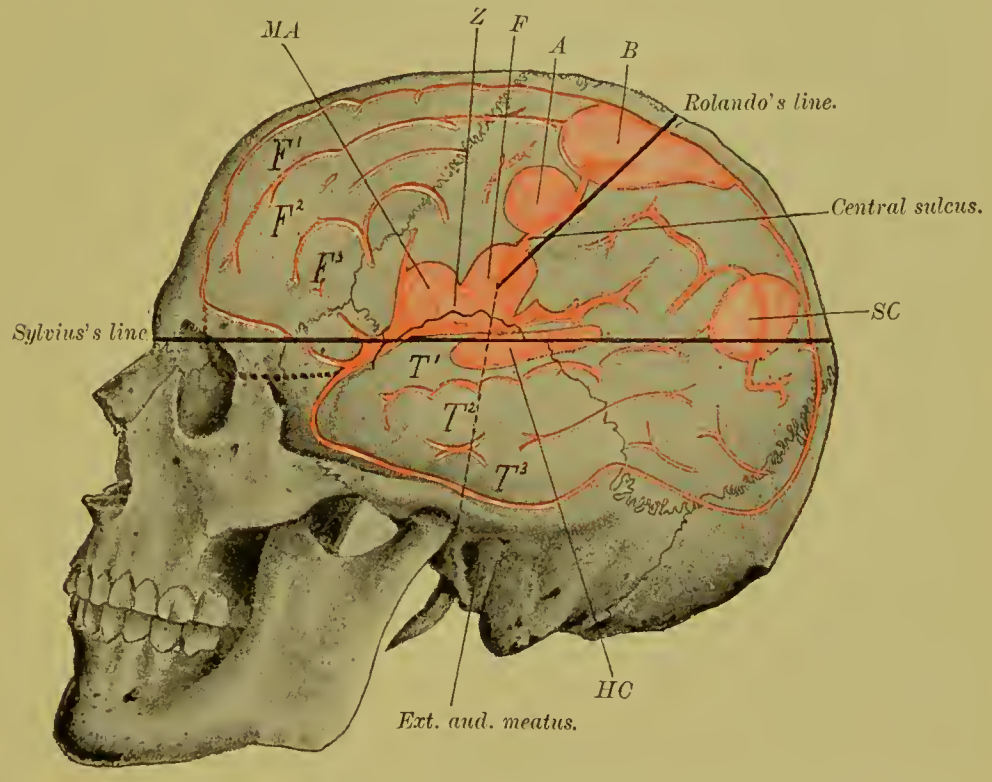


FIG. 161.—Topographical relations of the convolutions of the brain and of the cortical centers to the skull (after Poirier).[1]

MA, motor aphasia; *Z*, tongue; *F*, n. facialis; *A*, arm; *B*, leg; *SC*, sight-center; *HC*, hearing-center; F^1,F^2,F^3, first, second, third frontal convolutions; T^1, T^2, T^3, first, second, third temporo-sphenoidal convolutions.

Sensibility of the Cranium to Pressure.—This is ascertained by pressure with the finger or by gentle strokes with the tip of the finger or the percussion hammer. General sensibility to pressure occurs in nervousness, especially nervous pain in the head. We also sometimes meet with circumscribed sensibility to pressure in nervousness, likewise in hysteria; but sometimes the latter corresponds with a circumscribed meningitis, as this may be caused chiefly by tumors, abscess of the brain, etc. If other signs of a disease of this character are present, its topical diagnosis may be aided by palpation and percussion; by itself its results must be received with caution.

Regarding the significance of dilatation of the veins of the skull, see page 224.

Suppuration of the ear and nose (the latter seldom) plays an important part as causes of meningitis and abscess of the brain.

[1] Combination of several figures taken from his book, which is cited below.

The Spinal Column.—Form.—The significance of the expressions *scoliosis*, *kyphosis* (lateral and posterior curvature of the spine), and *kyphoscoliosis* have already been referred to on page 78. *Lordosis* is an abnormal curvature forward. If these curvatures are obtuse-angled, none of them have a deleterious effect upon the spinal cord, or at least only exceptionally. Acute-angled kyphosis (*gibbous*), as is usually caused by caries of the vertebræ, also by fracture of a vertebra, is of much greater importance, [causing] compression of the cord. It is to be remarked that in order to recognize slight lateral curvature it is desirable to mark the spines of the vertebræ, without moving the skin, with a blue crayon, and then to observe carefully the line that is thus formed. Any weakness or paralysis of the muscles of the spine on one or both sides may lead to secondary curvature of the spine, especially to scoliosis and lordosis.[1]

Diminished mobility of the spinal column, if it occurs with respect to the whole length in persons of mature years, is often not pathological. Complete general stiffness occurs also in arthritis deformans. If the stiffness is limited to a certain portion, while the rest of the vertebræ have free motion, this is of pathological significance (almost always due to caries, and here we sometimes have stiffness without curvature of the spine). Forcible bending is then generally painful. The spinal column is abnormally mobile when there is weakness or paralysis of its extensor or flexor muscles in young persons. This is especially marked in juvenile muscular atrophy, often in connection with habitual curvature.

Sensitiveness of the vertebral column to pressure (especially of the spines of the vertebræ) may have a great variety of significance. It may occur in palpable diseases, etc., especially in caries, but also with tumors of the vertebræ, of the spinal meninges, spinal meningitis, also tabes; but it may likewise occur with spinal irritation (particularly in the neck and between the shoulder-blades), as well as in hysteria, and here it may be excessive. We discover this sensibility by strong pressure or by striking the spines of the vertebræ. Often, but by no means always, there is at the same time painful sensibility when a hot sponge or the cathode of the galvanic current is passed over it.

Here, also, belongs the rigidity of the neck in meningitis, particularly basilar—an important sign of this disease; also the rigidity of the whole spinal column in spinal meningitis. With the former, by the contraction of the cervical extensors of the head, the latter is often bent back to a marked degree—"boring into the pillow." Backward bending of the vertebral column—*opisthotonos*—likewise occurs with attacks of tetanus; with epileptic, and especially hysterical, convulsions. With the latter, as the "arc de cercle," there are sometimes incredible distortions.

The anatomical relation of the cord to the spinal column is as follows: the cervical enlargement of the cord corresponds about with the third cervical or the first dorsal spine, the lumbar enlargement is about on the level with the ninth dorsal to the first lumbar vertebral spine; the *conus terminalis* begins at the first or second lumbar vertebra.

Puncture of the Vertebral Canal; Lumbar Puncture (Quincke).—This procedure was originally proposed for therapeutic purposes, but

[1] See still further regarding this under Function of the Muscles.

it has been more valuable for diagnosis, as has been shown by experience, than for treatment.

Method of Procedure.—The puncture is made under strict aseptic precautions. The patient is placed upon the side with the legs fully drawn up or in a bent sitting posture. The puncture is made with an aspirating syringe whose needle has an internal measurement of about 1 mm. The needle is introduced in the space below the arch of the second, third, or fourth lumbar vertebra, either in the middle line (children) or near it; in the adult puncturing close beside the spinal process, which projects into the space. The point of the needle is directed somewhat upward and, according to the age of the patient, pushed in from 2 cm. (maximum for children) to 6 mm. (maximum for adults). With a little practice it is easy to reach the subarachnoid space at the beginning of the cauda equina, when the cerebro-spinal fluid immediately flows out in drops or in a stream. The pressure and the amount of fluid vary within normal limits: sometimes also none at all escapes or the flow suddenly stops, probably in consequence of an obstructing filament of the cauda. The liquid which flows out is pure liquor cerebro-spinalis—*i. e.* clear like water, precipitates no fibrin, contains only a little albumin, but abundant salts, particularly sodium chlorid.

The use of this method for diagnostic purposes is greatly favored now-a-days by the well-known fact that the subarachnoidal spaces of the brain and spinal cord communicate with each other. For this reason the method has been employed for diagnosis of diseases of the interior of the skull even more than for diseases of the vertebral canal.

The *pressure under which the liquid flows out*, as has been mentioned, normally varies very much, and therefore it cannot be safely turned to account in diagnosis of increased cerebral pressure, as was to have been expected, in such cases as hydrocephalus and tumors, where the pressure has been found to be low.

On the other hand, the *quality of the liquid* may be turned to account in a different direction: in meningitis it contains fibrin, though also sometimes in tumors. The percentage of albumin is higher in meningitis than normal or in tumors, while in the latter sugar has been demonstrated. But other admixtures have much more weight: *blood* in rupture of a cerebral hemorrhage into the lateral ventricle; *pus* in all forms of *purulent meningitis*—also micro-organisms: in *meningitis tuberculosa*, in by far the majority of cases, there are found within the precipitating flocks of fibrin or after careful deposition by the centrifuge tubercle bacilli, which are demonstrable by the cover-glass method.[1] In some cases tuberculosis could be proved by culture. Besides, *in purulent meningitis* there occur strepto- and staphylococci, in *epidemic cerebro-spinal meningitis*, in rare instances the pneumococcus (Fränkel), more frequently a diplococcus very like the gonococcus, the meningococcus intracellularis, which in cerebro-spinal meningitis has also been found in the purulent secretion from the nose. It is stained in the same manner as the gonococcus.

The Peripheral Nerves and their Surroundings.—The nerves as the seat of disease come into consideration in all *peripheral paralyses*

[1] See p. 159, *ff.*

and in *neuralgias* (also, among others, in *reflex epilepsy*). In order directly to examine a nerve-trunk an exact knowledge of its course is necessary, and also of the organs that surround it from which an injurious effect upon the nerve may proceed.

By the examination of a nerve we learn its *anatomical condition:* any possible symmetrical thickening, with neuritis or perineuritis, unequal thickening or tumors in the nerve, with neuro-fibroma, neuroma; also any possible sensibility to pressure, as occurs with neuritis along the whole length of the diseased nerve, although this may be entirely absent. Finally, here belong the *sensitive points* in neuralgias.[1]

Moreover, a special examination must be made of certain points which, from any cause whatsoever, may easily be the starting-point of a disease of a peripheral nerve. These are—(*a*) those points where a nerve is especially exposed to traumatism because it lies near the surface of the body (especially if it at the same time lies over a bone). These situations essentially coincide, in part, with the electro-motor points to be mentioned later. Severe injuries, deep punctures, etc., of course, may destroy a nerve at any point. They are—(*b*) neighborhoods where a nerve may be exposed to injury from other organs. Here belongs compression by development of callus about the seat of fracture, especially of the bones of the extremities; also compression and sometimes inflammatory irritation from glandular tumors (axilla, neck, etc.), aneurysm, hernia (crural nerve); lesion of the facial nerve caused by caries of the petrous portion [of the temporal bone], etc. Indeed, in case of lesion of a peripheral nerve, we are frequently able to find the seat of the disease in this sense, but in every single case it must be looked for.

An extremely instructive case from the standpoint of diagnosis of the *locus morbi* was observed by Erb which was previously reported by the author. It was a case of ulnar neuritis resulting from exposure of the ulnar nerve from the fracture of the internal condyle of the humerus. The author has recently seen a similar case: both internal condyles of the humerus projected; the sulcus ulnaris was broad and shallow. In the first case there was a unilateral, in the second a bilateral, ulnar neuritis resulting from frequent injury to the nerve at its exposed point.

Examination of the Condition of the Mind.

In this section, which touches upon a territory foreign to this work —the mental state—we must, of course, limit ourselves to a brief mention of what is necessary in making a medical examination.

Mode of Examination.—An attentive observation of the behavior of the patient in bed, the expression of his countenance, his position, the reaction to external impressions, gives many disclosures regarding the faculty of perception and of his sensibility [or well-being]. By engaging the patient in conversation (Taking the Anamnesis, page 19) we are able to discover more regarding these points and to judge of the intellectual activity—*memory*, *imagination*, *possible delusions*, *the ability to think logically*. In testing the memory we take notice

[1] See p. 448.

of the recollection of things that are long past, as well as of more recent events, or of what has taken place during the present illness. The test of the power of thought and of the imagination is made by more or less simple arithmetical problems and by questions which are suitable to the social position and the occupation of the patient. We observe the great difference which various degrees of education produce in patients affected with the same disease, and we also take into consideration the age of the patient. We observe any possible diminution or increase of action, both instinctive, as the taking of food or sexual indulgences, and of actions with conscious purpose.

This expresses in general terms the course of the examination. To be sure, we shall very frequently be obliged, in order to recognize the first traces of a mental disorder, to take into consideration *whether the patient has changed in his nature or behavior.* Thus, for example, if a person becomes suddenly forgetful, careless, and disorderly, this will have quite a different significance than if he had always from his youth been so. Of course in regard to these things we must chiefly rely upon the statements of his relatives.

In what follows are given the explanation of the terms that have been adopted in the medical clinic, and the phenomena that accompany the several conditions:

Disturbances of consciousness are designated, according to their severity, as—*stupor*, also *somnolence* (sleepiness, lethargy, from which the patient can easily be awakened); *sopor*, in which the patient can only be awakened by decided appeals to his senses; *coma*, or complete loss of consciousness, in which the patient cannot be awakened in any way. The slightest degree of obtunded consciousness manifests itself in the scarcely noticeable trouble which it costs the patient to collect himself in order to answer a question or by his indifference with respect to being sick—a subjective sense of well-being. Further, there is an indication given by the sensibility to pain and the arbitrary or involuntary voidance of the stools and urine. In this respect the sensibility to pain often does not coincide with the other manifestations of consciousness.

Disturbance of consciousness occurs—in *acute infectious diseases*, especially in *typhoid fever*,[1] where the early manifestation of dulness has diagnostic value, but it may accompany any infectious disease, and may pass into deep coma; in *acute poisoning* of various kinds, especially from narcotics; as *uræmic*, *diabetic*, *carcinomatous coma*; as *epileptic*, *apoplectic coma*; in *meningitis*; in the most varying *diseases of the brain*, especially in tumors of the brain and its meninges. In the different forms of meningitis, however, consciousness may be retained for a remarkably long time. In tumors of the brain there is often for a long time a slight obscuration. It occurs also in *injuries* and *concussion of the cranium*; in large *hæmorrhages*; in *all chronic cachexias*, and at the end of life, at any rate in the last moments.

A patient who is in deep coma when he comes under the eye of the physician always causes great difficulty in diagnosis, the greatest when he can make no inquiry in regard to the patient. Systematic examination of the whole body is to be made—especially of the cra-

[1] See below.

nium for wounds; of the heart and blood-vessels; for evidences of apoplexy, meningitis; for signs of poisoning; of the urine, which is to be drawn with the catheter (for sugar, reaction for chlorid of iron, for albumin, casts; for certain poisons or as evidence of certain poisons, hemoglobin); lastly, of the stomach by evacuation (poisons).

Special Phenomena of Obtunded Consciousness.—**Delirium**—that is, talk and gesticulations arising from delusions—may follow any disturbance of consciousness, but it occurs especially frequently with acute infectious diseases; with severe cachexia, often as the end of life approaches; finally, as *delirium tremens seu potatorum* in chronic alcoholic poisoning. The latter manifests itself by talkativeness, restlessness, rapid alternations between passion and great anxiety, fear, hallucinations of sight (small black animals, especially mice, etc.), loss of sensibility to pain and cold; besides alcoholic trembling.[1]

The expression "muttering delirium" is used to designate a low murmuring with profound disturbance of consciousness. It is always a serious indication of great weakness and occurs particularly with typhoid fever.

Hysterical delirium forms a transition to the true psychoses, which cannot be treated here.

Spasms, Vomiting.—See the respective chapters.

Loss of consciousness, which quickly passes off, occurs as "syncope," "dizziness." This may be very benign, as in anemia and chlorosis, nervousness, great excitement, or severe pain. But it may have a serious significance—in elderly people as precursors of severe strokes of apoplexy; in kidney-disease as a sign of uremia; in disease of the heart and blood-vessels as angina pectoris, or as slight epileptic attacks (petit mal); lastly, it occurs in all possible chronic diseases of the brain, but especially in progressive paralysis. All of these conditions must be thought of when attacks of dizziness occur frequently in the same individual.

Dizziness, Vertigo.—In many respects this is to be looked upon as a slight, temporary loss of consciousness or connected with it.[2] But it only indicates a disturbance of the sense of equilibrium, and occurs as such most purely as a swimming of the eyes in diplopia[3] from deception regarding the location of objects in space and regarding the level of the floor. It also occurs in affections of the ear (*vertigo ab aure læsa*); in tumors of the brain, especially of the vermiform process of the cerebellum; in multiple sclerosis; with diseases of the stomach (*vertigo a stomacho læso*); in anemia; in cerebral neurasthenia; lastly, in chronic nicotin-poisoning.

Pathological depreciation of the power of the mind to perform its functions is designated as **imbecility.** It occurs in all gradations from moderate diminution in the perceptive faculties to a complete animal condition. Congenital imbecility is designated *idiocy*; when accompanied with certain physical manifestations, as *cretinism.* As an acquired condition it occurs as dementia senilis, also in organic diseases of the brain, especially tumors, apoplexy, multiple sclerosis; but also, as a temporary condition in convalescence from severe diseases, a

[1] See below. [2] See above. [3] See Eyes.

slight imbecility has been observed. Imbecility with delusions of greatness is a tolerably characteristic sign of progressive paralysis.

Of disturbances of volitional impulses are to be mentioned—*abulia* (hypochondria, drunkenness, indulgence in morphia); loss of desire for food—*anorexia;* certain forms of pathological excesses; *boulimia* (a morbidly great and unnatural appetite for eating all sorts of things), *nymphomania*, and *satyriasis* (abnormal sexual desires).

DISTURBANCES OF SENSIBILITY.

1. Sensitiveness to Peripheral Irritation.

The determination of the sensibility which a patient has for irritations applied from the periphery (by the physician) is made difficult by the fact that the estimation of them must rest with the patient, who is the subject of the experiment. Subjective sensibility, especially to pain, without doubt varies with individuals: with "torpid" persons and with the aged it is depreciated. Moreover, in a varying degree it is diminished with persons who are unconscious to the point of entire loss of sensation. Further, it will be influenced, when the irritation is slight, by the attentiveness of the person examined. The report of what is discovered in such examinations depends wholly upon the sincerity and good-will of the patient. We must always think of the possibility of *simulation* and *concealment* and the absence of favorable intention.

Very little weight must be given to the statements of the patient as to his capacity to feel. The most brief examination is best, as securing the most exact answers, for we very often meet with erroneous conceptions of the condition of the sensibility of the skin.

Whenever we are testing the sensibility it is advisable to *prevent the patient from seeing what we are doing.* If the disease is unilateral, it is desirable to make use of this circumstance to compare the diseased with the healthy side. As to how we are to guard against deception by simulation, see below.

Finally, it is most emphatically recommended to *employ the utmost similarity* possible *in the methods of making* one's *examinations*, for only in this way is it possible constantly to sharpen his own judgment. Moreover, every record of an examination should contain a statement of how the result was obtained.

Passing over the higher senses, the sensibility to peripheral irritation is divided into (*a*) cutaneous sensibility, (*b*) the so-called deep sensibility.

(*a*) **Cutaneous Sensibility.**—This, again, is divided into a number of qualities whose relation to each other and distinction one from the other are not yet entirely clear. We avoid any discussion of disputed points, and treat the qualities from the standpoint of clinical interest.

1. **The sense of touch,** sensibility to contact, is tested by gently touching the skin with the tip of the finger, the patient keeping his eyes closed, and whenever he feels the touch saying "Now"; it is better if he will also say "On the hand," or on the given finger, etc. Thus we approximately test the sense of locality.[1] And it is also recommended,

[1] See below.

in order to shorten the examination, to test the latter immediately more exactly by having the patient designate with the tip of the finger the spot that is touched. If he is able to do this, then his sense of touch and of locality is normal; if he cannot, there may be several reasons for his inability, as disturbance of the sense of touch and of locality, sometimes of the muscular sense, perception of position of the hand used in testing or pointing.[1] Then we must endeavor to separate the sense of touch from the sense of locality.

In many cases of slight disturbance the patient is able to feel the contact, but it is duller and different from what it is in normal places. Then we often obtain more exact information if we touch him with rough and soft materials and the like. In other cases this procedure is unnecessary.

2. **The local sense**, the power of localization, is tested by having the patient tell exactly where he has been touched. A healthy person can tell this with different degrees of accuracy according to the portion of the body which is touched. This about corresponds with the distances on the body which the related sense of space has been found to give.[2]

Testing the *sense of space* (only required when from any reasons the sensibility must be tested with the greatest exactness) is best done with Sieveking's esthesiometer: by means of two sliding points we are able to measure the shortest distance at which the two points can be recognized as two separate objects. In health the minimal distance, on the average, is as follows:

At the tip of the finger	2.5 to 5 mm.
In the palm of the hand	8 to 12 "
On the back of the hand	31 "
The forearm and the leg	about 40 "
The back	40 to 70 "
The upper arm and thigh	about 75 "

Analogous, although in its results not wholly corresponding to those of the above-mentioned method, is that of testing the *sensation of movements* (Leube): it relates to the power to distinguish points and the shortest lines that can be drawn upon the skin.

3. **The sense of pressure** residing in the skin is tested by the ability of the patient to determine the smallest differences between weights placed upon the skin. The limb must lie firmly, so that the muscular sense[3] is excluded. It is best to take blocks of wood of the same size (instead of metal), but made of different weight by being loaded with lead. The healthy person perceives differences of weight which are equal to about $\frac{1}{20}$ to $\frac{1}{30}$ of the absolute weight of the bodies employed. Partial paralysis of the sense of pressure is frequently observed, especially in tabes.

4. **The sense of heat and cold** is most quickly and simply tested by breathing and blowing upon the skin. Healthy persons distinguish the first from the second perfectly well. This method, however, is entirely unsatisfactory, because the finer disturbances of the sense of

[1] See below. [2] See p. 444. [3] See this.

cold and heat are not revealed by it. Somewhat more exact is the test made by means of two test-tubes filled with water at different temperatures. We must select a difference of temperature which we ourselves distinctly recognize, as, for instance, by passing the hand over them. If with one of these methods we find a disturbance of one of the two temperature-senses, then we can more exactly determine the degree of this disturbance by employing temperatures which vary still more; hence very low or very high (ice, hot water). At the same time we can thus determine the temperature at which cold- or heat-pain begins.

A finer test of the sense of heat is made by the aid of the thermesthesiometer. We recommend Nothnagel's—two cylindrical wooden vessels with metal bottoms, into each of which is dipped a thermometer to test the temperature of the water that is poured into them. In a very imperfect way we may make a substitute for this thermesthesiometer by using two reagent-glasses half filled with water. In these are placed thermometers surrounded by pledgets of wadding. The temperature of the glasses is varied by dipping them into vessels of cold or hot water. The thermesthesiometer enables us to determine exactly the fineness of the sensibility to heat and cold. The normal fineness of the sensibility to heat differs with the absolute height of the temperature which we select. Temperatures between 27° C. and 33° C. are most delicately distinguished. Here the recognizable differences in health average 0.5° C., except over the legs, where the number may be somewhat larger, and on the back, where it is about 1° C. On the cheeks it is about 0.25° C.

5. **Sensibility to Pain.**[1]—We recommend to test exclusively by pinching a fold of skin between two fingers, because in this way, with some practice — it depends very much upon the size of the fold of skin that is taken, and it is recommended always to press the rounded portion of the skin —we can best attain some uniformity in regard to the amount of irritation employed each time.[2] With patients who are unconscious it very often happens that the sensibility to pain is the only quality of sensation that is accessible to examination. When there is very decided unconsciousness we are made aware of it by the possible distortion of the countenance on account of pain or even a withdrawing of an extremity (not to be confounded with reflex of the skin).[3]

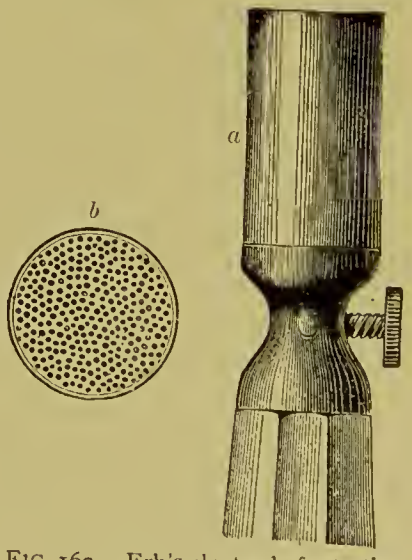


FIG. 162.—Erb's electrode for testing the sensibility of the skin.
a, tube of hard rubber; *b*, free surface of the electrode.

6. **Electric Sensibility.** — By the galvanic as well as the faradic current we can develop an objectively-visible as well as subjectively-painful sensibility of the skin. We confine ourselves to the description of the *farado-cutaneous sensibility.*

[1] Corresponding with the mode of procedure in making an examination, this is included here, although it properly belongs with Common Sensation (which see).

[2] Regarding pain caused by faradization, see below.

[3] See this.

It is best obtained by employing Erb's electrode for testing farado-cutaneous sensibility (made by Stöhrer in Leipzig), which is a cable of insulated copper wires cut at right angles with its axis. We mount this electrode upon the cathode of the opening current of a Dubois's induction-coil (the other electrode may stand anywhere upon the body) and notice the distance of rotation when the point of the skin under examination becomes sensitive (minimum of sensation), and also where it stands when pain is produced. Then, besides, we are to test the *galvanic resistance* at each point tested,[1] in order to have an approximate guide as to how strong a current, furnished by Dubois's apparatus, is exhausted by the resistance of the body (or of the skin) at the individual points; hence, how much of it is lost each time in producing the irritation of the skin. The following table gives the average figures of health as found by Erb, but we remark that the figures change according to the strength and construction of the induction apparatus employed, and also that the deviation of the needle (for testing the galvanic resistance) was attached to an old galvanometer without absolute divisions. For both of these reasons the relation of the figures to each other, rather than the absolute variation of the needle indicated by them, is of value:

Points of resistance.	Minimum.	Pain.	Deviation of the needle with 8 elements; conduction resistance 150.
Cheeks	200–220	130	26°
Neck	170–200	120	22°
Upper arm	200	120	21°
Forearm	190	115	18°
Back of the hand	175	110	15°
Tip of the finger	125	90	2°
Abdomen	190	120	20°
Thigh	180	115	21°
Lower leg	170	110	19°
Back of the foot	175	110	10°
Sole of the foot	110	80	5°

The method is further liable to error, regarding which we cannot speak here.

Farado-cutaneous sensibility does not go entirely parallel with any other quality of sensibility. Most frequently, but not always, the sensations of pain produced by pinching, and the minimal sensations of pain produced by the faradic current, correspond with each other (this is especially the case in tabes). The method has not yet been sufficiently studied to be of independent diagnostic significance, and particularly to have a value for special diagnosis. Its application is chiefly to be recommended in unilateral slight disturbance of sensibility, from the possibility of making a comparison with the sound side, which cannot be quite certainly established when there is normal irritability of the skin.

Regarding *stereognosis*, see page 446.

Now, if by testing the sensibility we find it diminished, we speak of *hypesthesia*, often incorrectly spoken of as anesthesia. If none is found—that is, if the strong or maximal irritation employed, which

[1] See under Electrical Examination for Motility.

is always to be stated as accurately as possible, meets with no response —then we speak of loss of sensibility, or *anesthesia*. Heightened sensibility is *hyperesthesia*, or sensibility to variations of temperature and to pain. In many cases, especially in diseases of the peripheral nerves, the *sensibility* is *equally altered* in all its qualities; in others, and especially in diseases of the spinal cord, in cerebral anesthesia, and not infrequently in hysteria, there exists a *partial paralysis of sensibility*. Of this the most frequent form is the diminution or absence of sensibility to pain—*analgesia*.

When *sensibility* is *slowly conducted* ("*delayed sensibility*") it is recognized by requiring the patient, with his eyes closed, to call out "Now" the instant he has a sensation. Sometimes the pause can be measured by seconds (ten seconds and more). This phenomenon is most frequently observed with reference to pain, as in tabes and in peripheral paralysis. If we take hold of the patient and immediately pinch him, he will often call out "Now" twice, because he felt the touch, and then, later, the pinch: there is *double sensibility*. For this reason it is best to take up the skin first without pressing it, and then suddenly to pinch it.

Gradual increase of sensibility to pain, when inflicted, so that just at the moment of being pinched it is inconsiderable, and later the pain increases markedly, appears by its phenomena and occurrence to be related to delayed communication of the pain.

Perverse sensibility to changes of temperature (Strümpell) consists in cold being experienced as heat. According to our recent views of the complete opposition of the sensibility to heat and the sensibility to cold, this disturbance is not, as yet, explicable. Yet it has an analogy in those rare anomalies of sensibility where a gentle touch is felt as cold.

After-sensibility (Naunyn) is a term used to describe a pain that when first inflicted immediately subsides, but for some time after returns, and, indeed, with increase of intensity.

Polyesthesia (Fischer): when one point of the esthesiometer is placed upon the surface, it feels as if there were two.

Allochiria (Obersteiner): when the right extremity is touched it is referred to the left, and *vice versâ*, as in tabes, myelitis, hysteria, multiple sclerosis.

Local Manifestations of Disturbed Sensibility.—Of course these are to be determined as accurately as possible. This is very easily done when the disturbance of sensibility is sharply bounded; however, not infrequently the region of disturbed sensibility of the skin passes very gradually and indistinctly into the normal portion. Total anesthesia is a curiosity. Unilateral anesthesia or *hemianesthesia*, not passing beyond the middle line of the body, sometimes affecting the head, trunk, and extremities (including the mucous membrane) in a similar way, occurs with certain deposits in the internal capsule (in the posterior third of its posterior limb) and in hysteria. In the latter, and (it is said) also in the first case, there is simultaneously exact unilateral disturbance of all the higher senses. *Para-anesthesia* is anesthesia of both lower or both upper limbs. A zone of disturbed sensibility, a territory of any extent, may exist in all imaginable parts of the body.

If it is small, it may easily be overlooked, unless the search for it is very carefully made: this is particularly apt to be the case in the extremities. Here especially (but also on the trunk) we must carefully determine whether the anesthesia corresponds with the region of distribution of a cutaneous nerve or of a mixed nerve-trunk,[1] or whether it is not confined to such a territory—that is, "diffuse" or "washed out." In the first case it would indicate an isolated disease of that particular nerve. Anesthesia (analgesia) affecting an extremity which is limited to the portion distributed about a joint (say, as far as the wrist or up as far as the elbow-joint, etc.) has been met with in certain functional neuroses, especially of the so-called hystero-traumatic neuroses of the French.

It may happen—indeed, it very frequently does—that an anesthetic territory does not really comprise the limits of a nerve of the extremities, but the inner half of it is wanting. Thus in a radial paralysis there may be an anesthetic zone (easily overlooked) confined to a small part of the dorsal side of the forearm. This results, either because the nerve is not interrupted throughout its whole transverse section, or because we have that very puzzling phenomenon, the "vicarious" participation of a neighboring nerve.

(*b*) **Deep Sensibility.**—This is divided into the less important categories of *the dynamic sense*, the *sensation of spasm* of the muscles, and the important so-called *muscular sense*, which is a generic name for a series of sensations.

Dynamic sense is the capacity to recognize the weight or the difference of weight between different bodies which one lifts. It may be exactly tested only with the upper extremities, and even here it is not wholly separable from the pressure-sense of the skin. Different weights are placed in a cloth sling pulled over the hand on to the wrist. A healthy person will recognize differences of one-fortieth [of one kilogram].

Sensation of spasm is the unpleasant sensation or pain which is experienced in very strong contraction of the muscles, as in cramp in the calf of the leg or strong faradic muscular stimulus with anesthesia of the skin.

Muscular Sense.—*Perception of Movement.*—By this we understand the ability to recognize, with the eyes closed, the position a limb is in (*conception of localization*) and the active and passive motions of a limb. It is due to the sensibility of the muscles, joints, and their ligaments, by the feeling of varying tension of the skin in flexion and extension of a joint, by the impressions of touch which come from portions of skin being in contact, as in the axilla and elsewhere. In a rude way we test the sensation of location and of motion in the arm (with the eyes closed) in persons with unilateral disease as follows: we place the diseased arm in different positions, and have the patient with the sound hand take hold of the wrist of the diseased arm. The same method may be employed in unilateral disease of the leg. Besides, it is well, when there is disease of the legs and bilateral affection of the arms, to have the patient describe the positions in which they are placed or the passive motions of the joint that are made. We

[1] See p. 448.

can also have the patient describe and represent numbers in the air with his hands.

By *perception of movement* Goldschneider understands the perception of passive movements only. He has studied this very carefully, and has particularly examined the physiological limits of its delicacy. For this purpose he has devised a "movement measure," constructed on the principle that the force exerted by the passive motion makes an angle corresponding to a departure from a vertical line. In this way he has determined the physiological limits, which are expressed by an angle. Some of his findings are here given, which show approximately the delicacy of this perception:

Shoulder-joint	0.3°–0.6°
Elbow-joint	0.5°–0.8°
Wrist-joint	0.3°–0.6°
Knee-joint	0.5°–1.0°

The first figure, for example, indicates that in healthy persons a movement at the shoulder-joint so slight that the humerus changes its relation to the extent of only 0.3°–0.6° is distinctly perceived. It matters not what the point of departure is, so long as there is no discomfort, tension, etc.; nor is the direction of the passive motion of importance. The test of the perception of passive motion is of course to be made with the eyes closed, so as to avoid as far as possible the sensibility excited in the skin by the seizure or motion of the limb by the examiner. The proximal part of the tested limb must sometimes be held firmly. The full account of these studies is given in *Berliner klin. Wochenschrift*, 1890, p. 322 *f.*

Romberg's Symptom.—The patient places his feet close together; as soon as he closes his eyes he begins to reel; sometimes he may fall. The phenomenon is dependent upon anesthesia of the soles of the feet and disturbance of the muscular sense of the legs, which is no doubt increased by the existing ataxia,[1] because in this condition the motions to correct the swinging are too violent; this is especially characteristic of tabes dorsalis. [But something of this symptom may be present in health, owing to the lack of vision to correct incipient lateral movements. This may be made clear by closing the eyes and then attempting to stand on one foot.]

A finer test of the muscular sense may be made by placing before the patient a table with numbered squares like a chess-board, each square measuring about 10 cm. on a side, and having him point them out with the eyes open until he has them all in his head, and then with closed eyes to touch them with the hand, or, on the other hand, the patient moves his hand about the squares and names the fields as he comes to them. With the legs the same test may be made with cubes measuring 10 cm. on the side, placed one on top of another and then side by side. This test, however, requires a certain degree of intelligence on the part of the patient.

Conception of space ("finding one's position in space") can be tested by placing substances of different thicknesses between his thumb and forefinger to ascertain the smallest perceptible differences of thickness.

[1] See this.

In testing the conceptions of active motions we see that it is very much disturbed in paralysis, ataxia, and chorea.[1]

The Knowledge of Form (Stereognosis).—We recognize the form of bodies partly by the sensibility of the skin and partly by deep sensibility. The former is employed more for very small bodies (which can be grasped with the hand; here, indeed, the hand is the chief means), the latter more for large substances. Thus far, only the recognition of small bodies has been sought, especially in an exact way by Hoffmann.

Hoffmann's Test.—To make this test he selected a ball, half-ball, segment of a ball, a cone, a die, a three-cornered pyramid, a regular octahedron, and a dodecahedron—all of a size for the hand to grasp. He chiefly tested the hand of persons in health and sick people as regards their ability to recognize these bodies (to which popular names were given).

Hoffmann and others have found that the recognition of small bodies was principally made by the skin and sense of space and of pressure of the skin, and to a less degree by the sense of motion in the joints and the power of determining the location in space. Also, that the active to-and-fro motion of the body in the hand for a different reason comes into consideration: if the active motion is wanting, then the stereognosis is hindered, but not abolished.

At present the examination of stereognosis has no independent value; testing the separate qualities of sensation is superior to it. According to our experience, the most important result of Hoffmann's examination is the knowledge that the separate factors of stereognosis may very perfectly act one for another when there are pathological disturbances.

2. Sensible Phenomena of Irritation and Pain from Pressure upon Nerves.

1. Paresthesia.—This occurs as a subjective sensation of touch, like fur, creeping of ants, creeping of insects, falling asleep; also as a subjective sensation of pain, as a fine stinging or pricking, and also a severe pain; lastly, as a subjective sensation of cold and heat or painful burning.

The so-called *feeling of constriction*, which occurs most frequently upon the trunk in the region of the thoracic vertebræ, especially in tabes, but also in local disease of the spinal cord and its meninges, belongs here. Generally it is a sensation of tension, but it also occurs in all stages of transition to genuine neuralgic pains when it is deeply located.[2]

2. Spontaneous Pain.—Headache (Cephalalgia).—This, according to the manner of its occurrence as well as its significance, may be extremely varied in its character. Its chief forms are—

(*a*) Headache produced by palpable disease of the meninges in the different forms of meningitis—in all those diseases of the cranium and the brain which accompany meningitis. If the affection is circumscribed, the headache may likewise be so, and it then sometimes indicates the location of the disease; but also often enough in this case it is not located.

[1] Regarding these, see below.

[2] See below.

Related with this are the nocturnal headaches of syphilis.

(*b*) The headache of neurasthenia is quite various in its onset. Sometimes it appears as a painful pressure in the head, sometimes as extremely severe pain; again, it is diffuse, then localized, especially at the crown of the head. There is the hysterical headache, not infrequently circumscribed at the crown (clavus hystericus).

(*c*) Migraine. This is generally an unilateral headache occurring with pauses of extremely varied duration, which is associated with disturbances of the stomach, scintillations,[1] tinnitus aurium, dilatation or contraction of the pupil of the affected side, etc. The condition is idiopathic or symptomatic, especially in tabes, tumors of the brain; also sometimes in diseases of the nose, etc.

(*d*) Neuralgia in the head.[2]

(*e*) Toxic headache occurs particularly in chronic poisoning with lead, mercury, alcohol, nicotin. Here also belongs the headache of uremia.

(*f*) There is a headache which occurs in the beginning and during the course of acute infectious diseases, especially intense and long continued in typhoid fever.

(*g*) Anemic headache—headache with gastric dyspepsia; abdominal diseases of all kinds, especially diseases of the female sexual organs.

(*h*) The so-called habitual headache. Often there is an hereditary disposition to headache, which occurs with exertion, excitement, bodily disturbance, as catching cold, etc., and the disposition generally lasts during the greater part of one's life.

Pain in the spine may concern the vertebræ, as in chronic rheumatism, arthritis deformans, caries; the spinal muscles, as in muscular rheumatism; the spinal cord or its meninges, especially in meningitis and in tabes with tumors. But it occurs very frequently, and is especially torturing, in neurasthenia and spinal irritation.[3]

Neuralgia.—This is generally a severe paroxysmal pain occurring in the region of one or more distinct nerves. It may be idiopathic or result from catching cold, but it may also be symptomatic, with the greatest variety of significance. The principal varieties of neuralgia are those produced by mechanical irritation (pressure of a tumor, aneurysm, periostitis, etc.); sequela of inflammation of the affected nerve; neuralgia dependent upon infectious or toxic influences (malaria, syphilis, lead, mercury, nicotin, etc.), or accompanying constitutional diseases, as diabetes, gout, phthisis. In every neuralgia we are to keep in mind the whole course of the affected nerve, and consider where and how it may be injured, and how such a local injury may directly or indirectly be discovered.

Of special importance are the *neuralgic, lightning-like, lancinating pains* in the initial stage of tabes dorsalis. They occur very much more frequently in the lower extremities and the trunk in the region of the intercostal nerves, and now-a-days are not infrequently confounded with rheumatism. Also in the beginning of multiple neuritis there are neuralgic pains, although generally of moderate intensity.

We have previously mentioned the *pain produced by pressure* upon

[1] See the Eye. [2] See below.

[3] See also what was previously said regarding the vertebræ.

the head and upon the vertebræ. The peripheral nerve is sensitive to pressure in neuritis whenever this is accompanied by actual inflammatory phenomena in the nerve or there is perineuritis. Very frequently there is especially pronounced tenderness of the nerve during an attack of neuralgia, but also often, although to a slighter degree, in the intervals. This tenderness is very great at certain points of the nerves, especially where the nerve can be pressed against the bone (*Valleix's points*) [points douloureux].

Tenderness and spontaneous pain in the joints, without anatomical changes and generally very changeable in severity, are characteristic of articular neuralgia.

Distribution of the Sensory Cutaneous Nerves.

It is recommended that the accompanying figures [Figs. 163 and 164] be studied, in connection with which we will draw attention to a few points which seem to us to be especially important:

1. The Nerves of the Head.—It is to be noticed that the nerve V_1 also supplies the conjunctiva and a portion of the mucous membrane

FIG. 163.



FIG. 164.



FIGS. 163 and 164.—Distribution of the cutaneous sensitive nerves upon the head (Seeligmüller).

oma, *omi*, N. occipit. maj. and minor (from the N. cervical, II. and III.); *am*, N. auricular. magn. (from N. cervic. III.); *cs*, N. cervical, superfic. (from N. cervic. III.); V_1, first branch of the fifth (*so*, N. supraorbit.; *st*, N., supratrochl.; *it*, N. infratrochl.; *e*, N. ethmoid; *l*, N. lachrymal); V_2, second branch of the fifth (*sm*, N. subcutan. malæ seu zygomaticus); V_3, third branch of fifth (*at*, N. auriculo-tempor.; *b*, N. buccinator.; *m*, N. mental); *B*, posterior branches of the cervical nerves.

of the nose; further, that when it is paralyzed we observe severe inflammation and ulceration of the eye (ophthalmia neuroparalytica), which until recently were regarded by most persons as arising from lesions, as by dust, etc., which were not warded off because they had not been seen. The author inclines to the old view that the disturbance of nutrition forms the starting-point of the trouble. Nerve V_2 supplies the mucous membrane of the superior maxilla, a part of the gums and of the nose, the upper teeth, and the chorda [tympani] accompanies its

trunk: hence sometimes there is disturbance of the taste at the anterior portion [two-thirds] of the tongue. Nerve V_3 supplies a portion of the tongue and the mucous membrane of the cheek and presides over the secretion of saliva. It contains motor fibers, of which the



FIG. 165.

V_1, V_2, V_3, 1st, 2d, and 3d branches of the trigeminus; *C*, cervical nerves; *B*, brachial nerves; *ax*, N. axillaris; *cmd*, N. cut. medialis; *cm*, N. cut. medius; *cl*, N. cut. lateralis; *IC*, intercostal nerves; *ra*, rami anteriores; *ri*, rami laterales; *L*, lumbal nerves; *ih*, N. ileo-hypogastricus; *ii*, N. ilio-inguinalis; *li*, N. lumbo-inguinalis; *se*, N. spermatic ext.; *cl*, N. cutan. lateralis; *cr*, N. cruralis; *obt*, N. obturator; *sc*, Nn. scrotales; *dp*, N. dorsalis penis; *cp*, N. cutan. post. (the last three sacral plexus).

most important are those distributed to the muscles of mastication (masseter, temporalis, pterygoideus ext. et int.).

2. Nerves of the Neck and Trunk.—These do not require any further explanation (compare Fig. 165).

3. Nerves of the Shoulder, Arm, and Hand.—Here we are especially to note the smallness of the cutaneous filaments of the radial nerve that supply the dorsal side of the forearm. Anesthesia here may

easily be overlooked. It is to be remarked also that the distribution of the cutaneous nerves to the fingers, and also to the hand, is subject to some changes, so that slight variations from the arrangement usually described ought not to lead to mistake. Lastly, very often on examination of a peripheral paralysis it is found that the extension of the sensory disturbance lags behind that of the motor. The phenomenon is largely explained by a vicarious participation of neighboring cutaneous nerves in a portion of the territory affected (notwithstanding the

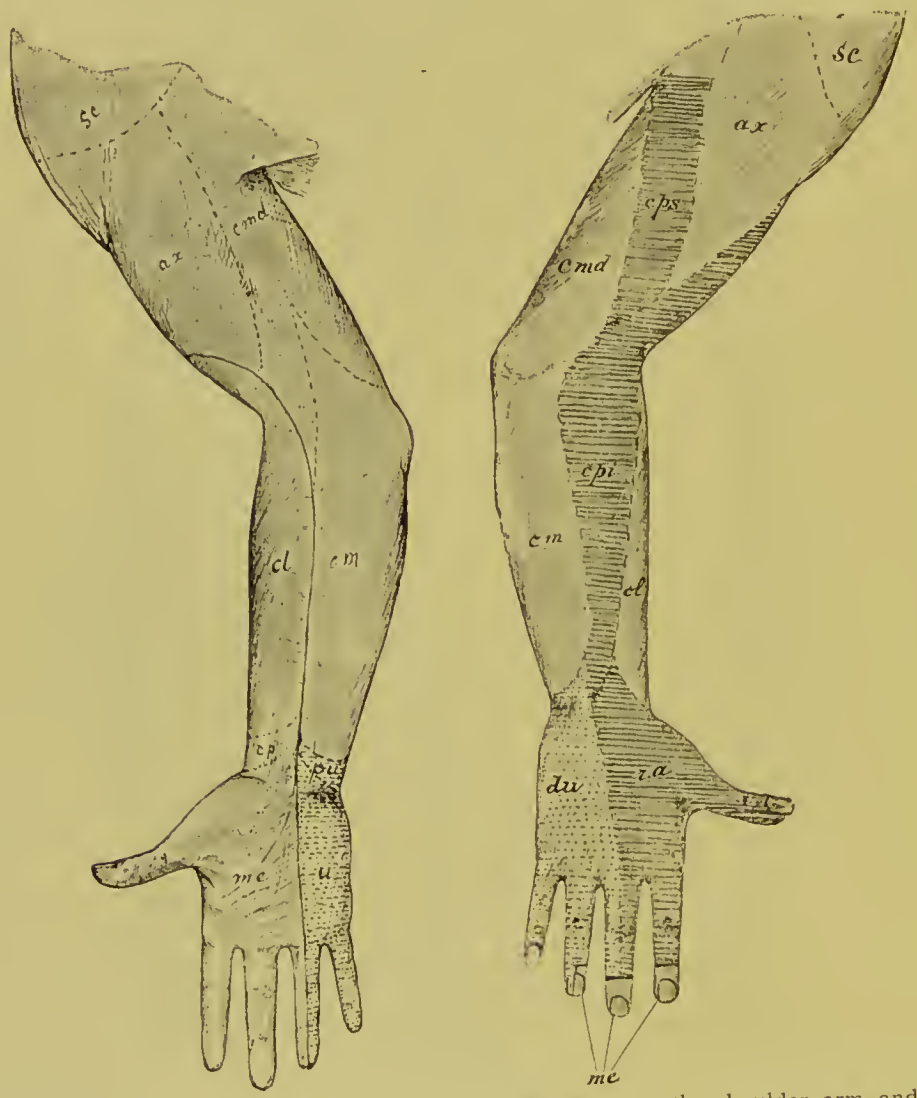


FIG. 166 *a* and *b*.—Distribution of the cutaneous nerves to the shoulder, arm, and hand (Henle). The region of the N. radial. is represented by the unbroken hatched lines, that of the N. ulnaris by the broken hatched lines.

a, anterior, *b*, posterior surface; *sc*, Nn. suprascapular (plexus cervicalis); *ax*, chief branch of N. axillar; *cps*, *cpi*, Nn. cutanei post. sup. and inf. (from N. radialis); *ra*, terminal branches of N. radial; *cm*, *cl*, Nn. cutanei medius (also to the plexus) and lateralis (chiefly to the N. medianus); *cp*, N. cutan. palmar., N. rad.; *cmd*, N. cutan. medialis; *me*, N. medianus; *u*, N. ulnaris; *cpu*, N. cutan. palm. ulnaris.

many investigations regarding its existence, this idea of vicarious action has not yet been as clearly explained as is desirable).

Paralysis of the brachial plexus at Erb's point[1] sometimes causes anesthesia in the region of the median nerve. Paralysis from compres-

[1] See Electrical Examination.

sion of the radial [musculo-spiral] at the point where it passes around [the humerus] causes sensory disturbance only at the hand,[1] because the posterior cutaneous nerves [internal, supplying the posterior and internal aspects of the arm as far as the elbow; and external, arising from the nerve on the outer border of the arm, is distributed to the

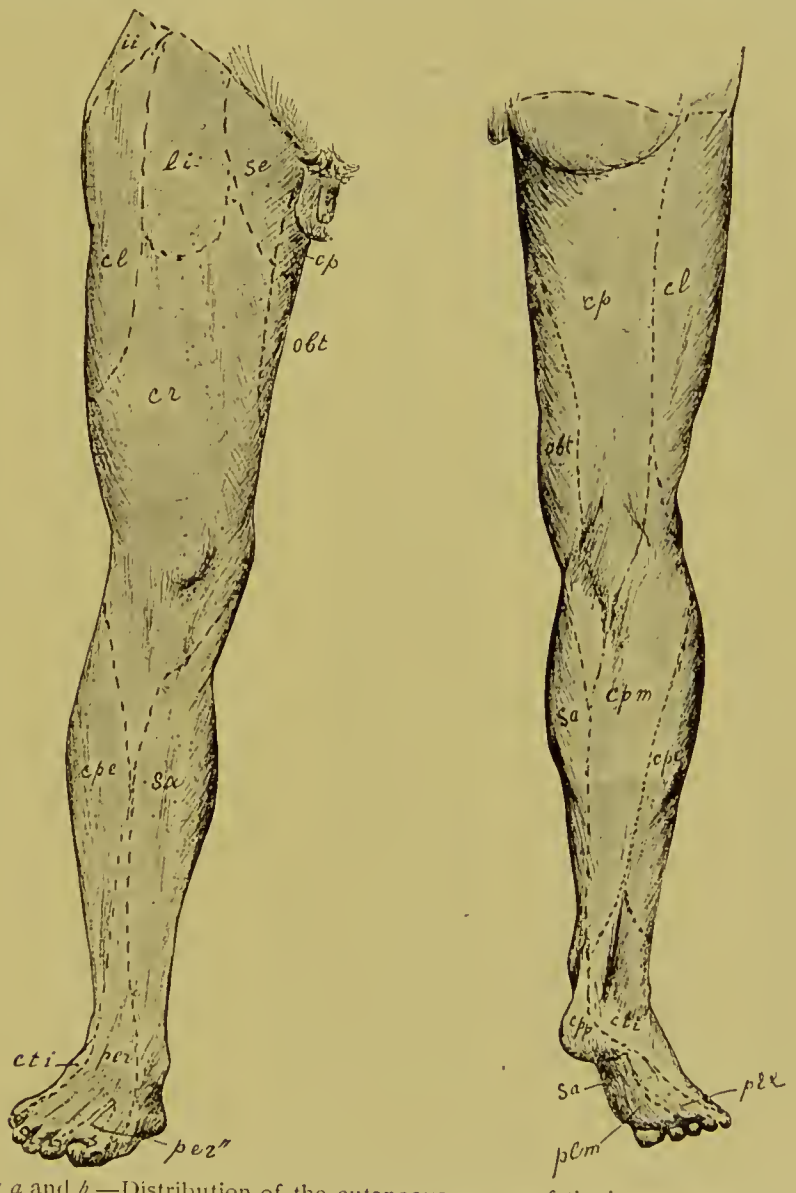


FIG. 167 *a* and *b*.—Distribution of the cutaneous nerves of the lower extremity (Henle).

ii, N. ileo-inguinal (plex. lumb.); *li*, N. lumbo-inguinal (to the genito-crural. plex. lumbal.); *se*, N. spermat. ext. (to the genito-crural.); *cp*, N. cutan. post. (plex. ischiad.); *cl*, N. cutan. lateral. (plex. lumb.); *cr*, N. cruralis (plex. lumbal.); *obt*, N. obturator. (plex. lumb.); *sa*, N. saphen. (plex. lumbal.); *cpe*, N. commun. peron. (N. peron. tibial.); *cti*, N. commun. tibial.; *per'*, *per''*, N. peronæi ram. superfic. et prof.; *cpm*, N. cutan. post. med. (plex. ischiad.); *cpp*, N. cut. plant. propr. (N. tib.); *plm*, *pll*, N. plantar. medial. et lateral. (N. tib.).

back of the forearm] are given off above the point of circumflexion. On the other hand, compression of the radial in the axilla (crutch-paralysis) often causes anesthesia of the forearm.

4. Nerves of the Lower Extremities.—(See the accompanying figure, Fig. 167 *a* and *b*).

[1] See Electrical Examination.

DISTURBANCES OF MOTILITY.

In this connection we consider not alone the disturbances of muscular action in the strict sense, but also the manifestations as respects *tonus* and the *nutrition of the muscles*, the co-ordination of their actions, their electrical and mechanical irritability, and their reflex manifestations.

1. Paralysis.

By paralysis of a voluntary muscle we understand a condition in which, by the action of the will, it can only to a diminished extent, or cannot at all, be made to contract. If there is complete absence of voluntary contraction, we call the condition *paralysis;* if the power of voluntary contraction is only diminished, it is called *parcsis.* Paralysis is the result of some anomaly of the muscular nervous system or of its motor terminal apparatus.

The loss of motion due to *stiffness of the joint* has nothing to do with paralysis. Such inability to move a joint is especially frequent in the extremities, and may lead the inexperienced into error. If there is simultaneous stiffness of the joint and paralysis, it may be extremely difficult to determine the existence of the latter. Diminution of power of motion caused by *pain* has nothing to do with paralysis when there is only a want of self-control on the part of the patient. However, very severe pain may cause a local restriction of movement, which is, in fact, to be considered as a paralysis.

Phenomena of Paralysis; Methods of Examination. — Paralysis is recognized by the complete absence of the power of motion in the sense of action of the affected muscles, and, as regards the muscle itself, by the absence of contraction that can be seen or felt. An extensive paralysis, if it causes the muscles to be lax,[1] produces a characteristic atonic behavior of the affected limb: if we raise it and then let go, it falls—an important symptom of loss of consciousness. As regards those muscles (and there are many such) whose failure does not in a very noticeable degree affect the motion of a limb because their actions are replaced by others, we *recognize the paralysis* by observing and feeling the muscles during active movements of the joint which would likely call them into action; among such belongs the supinator longus. *Parcsis is recognized* by the diminution of "native vigor" when resistance is called for; and also, supposing the joint to be free and an absence of tension on the part of the antagonizing muscles,[2] by diminished freedom and rapidity of motion. Again, we sometimes resort to an attentive examination and careful feeling of the body of the muscle. On the other hand, we may be deceived by the statement of the patient that he has a feeling of great lassitude.

For the examination of individual muscles, see page 492.

Extent of Paralysis.—Paralysis of one-half of the body, with or without paralysis of the corresponding side of the face, is called *hemiplegia.* Paralysis of one side of the face, of an arm, a leg, is called *monoplegia facialis, brachialis, seu cruralis.* We also speak

[1] See below.

[2] See Tonus of Muscles.

of monoplegia brachio-facialis. *Paraplegia inferior* is paralysis of both legs; *paraplegia superior*, of both arms. *Hemiplegia cruciata* signifies paralysis of the arm of one side and the leg of the opposite side; *hemiplegia alternans*, or likewise *cruciata*, paralysis of an extremity of one side and of the facial or oculo-motorius of the other side.

The extent of the paralysis is an extremely important aid in diagnosis, as follows from the anatomical remarks made at the opening of this section. For anatomical diagnosis see further, below.

2. Disturbance of the Nutrition Tone of the Muscles.

Nutrition shows manifest differences that are very striking, and of the highest diagnostic importance. It is determined by the volume of the muscle and by its electrical behavior.[1]

More or less symmetrical diminution in the volume of the muscles of a portion of the limb is designated as *diffuse atrophy;* when it affects a single muscle, as *circumscribed atrophy*. A corresponding increase in the volume is called *hypertrophy* or pseudo-hypertrophy.[2] The existence of atrophy and its extent are determined by inspection and palpation; if possible also by measuring. Whenever one side alone is affected, we are always to compare it with the healthy side. Requiring the patient to make active motion, by which the muscle under examination is made to contract, or which causes contraction in the surrounding muscles, often makes the impression much clearer. We can easily combine testing the strength with the examination of the state of nutrition.

The *volume of an extremity* is measured with the tape-measure while the limb is extended at rest (both arms and both legs are to be in exactly the same position), and it is best done at certain points of election.

We measure the upper arm at the point of its greatest circumference; the forearm, 2 to 3 cm. below the lower margin of the inner condyle of the humerus; the thigh, 15 cm. above the upper edge of the patella; the calf of the leg, at its greatest circumference.

Thus in measuring the forearm and the thigh we must first fix the point where we are going to take the measure, and mark it with a blue pencil.

Atrophy is divided into the following varieties, which are to be very sharply distinguished from each other:

(*a*) **Atrophy of Inactivity.**—This consists of a diminution in the volume of the muscles, which is very slight, and which very slowly develops in the course of months of inactivity. Almost without exception it supervenes in cases of paralysis, and also in any long-continued inaction of the muscles, as in surgical diseases which require the limb to be kept at rest. In this form of atrophy, as will be shown later, the electrical sensibility of the muscles is qualitatively unchanged.

(*b*) **Degenerative atrophy**, with the so-called atrophic paralysis. This quickly leads to a high degree of atrophy of the affected muscles,

[1] See Electrical Examination.

[2] See this below.

and to a qualitative change in their electrical sensibility—the *reaction of degeneration.*[1] This degenerative atrophy only occurs if the center which presides over the nutrition of the muscle, hence that portion of the gray matter of the anterior horn corresponding to the affected muscle, is disturbed or is separated from the muscle; therefore in all primary and secondary diseases of the anterior horns, in local separations or interruptions of the connection with the anterior roots or peripheral nerves, in peripheral neuritis.

Here belong: poliomyelitis acuta, subacuta, chronica; progressive muscular atrophy of spinal origin; amyotrophic lateral sclerosis; all processes within and of the spinal cord which destroy the gray substance (tumors, hemorrhages, softening); compression of the anterior roots and the peripheral nerves; traumatic complete separation; severe contusion; pressure-necrosis of these; and all forms of acute and slow degeneration or degenerative neuritis.

Also, it will be understood that the motor nerves below the seat of the lesion, as far as to the muscle, atrophy.[2]

On the other hand, *degenerative atrophy is wanting* in all paralyses which are due to a disease of a motor tract above the anterior horn-ganglia—that is, in the *pyramidal tract of the spinal cord, of the brain,* and *in the cortex of the brain.* Therefore in these cases we only have the atrophy of inactivity. Moreover, degenerative atrophy is wanting in *paralyses of myopathic origin*[1] and in *functional paralyses.*

Nevertheless, degenerative atrophy in many diseases occurs in such a way as to cause great clinical difficulties: the rapid (developing within fourteen days) diminution in the volume of a muscle of course can only occur when the whole of the affected muscle or a large compact portion of it is suddenly, at an approximately definite time, completely paralyzed by disease of the anterior horn or of a peripheral nerve (poliomyelitis acuta, section of a nerve, rheumatic facial paralysis, etc.). A disease developing slowly in the course of weeks and months causes slowly progressive atrophy, at first disseminated in the separate muscular fibers, only gradually becoming general. There are also difficulties in determining the *reaction of degeneration* in such slowly extending degenerative atrophy.[1] We have the greatest difficulty in making out degenerative atrophy when the disease is a disseminated one, in which bundles of muscular fiber that are still normal are distributed everywhere between diseased bundles.[3]

It is to be remarked that all cachexias cause general atrophy as well as atrophy of the muscles. But it is worthy of still further note that under the influence of a general atrophy the paralyzed muscles often become excessively atrophied, even when the atrophy is not a degenerative one. In cases of myelitis transversa and simple atrophy of inactivity of the legs, when there comes to be a general atrophy, we have often seen the legs become extremely atrophied, quite out of proportion to the volume of the arms. But there is no reaction of degeneration, and this fact furnishes diagnostic assistance.

It is often extremely difficult for the beginner to form a conception

[1] See below. [2] See above, p. 419, and also under Electrical Examination.
[3] Regarding this see further, under Electrical Examination.

of the behavior of the anterior gray columns when there is disease of a transverse section of the spinal cord, and to answer the question in connection with it, What sort of paralysis will result from such disease? For this reason two examples are presented:

In a severe contusion of the prominence of the neck (fracture of a cervical vertebra, for instance) it may happen that the whole section of the anterior gray columns, which innervates the arms, is disturbed, and that simultaneously the pyramidal-tract fibers for the muscles of both legs are unbroken (at *H* in the figure): there follows a degenerative atrophic paralysis of the arms and a non-atrophic, "simple" (spastic[1]) paralysis of the legs. The pyramidal-tract fibers of the latter degenerate as far as the lumbar portions of the cord (as far as *L*), but the degeneration stops here: the anterior-horn ganglia remain normal, and hence the peripheral nerve and muscle also.

A myelitis transversa of the dorsal portion of the cord interrupts the pyramidal tracts to the legs; these become simply (spastically) paralyzed; a myelitis transversa of the lumbar portion of the cord disturbs the anterior-horn ganglia of the legs: these are affected with atrophic paralysis.

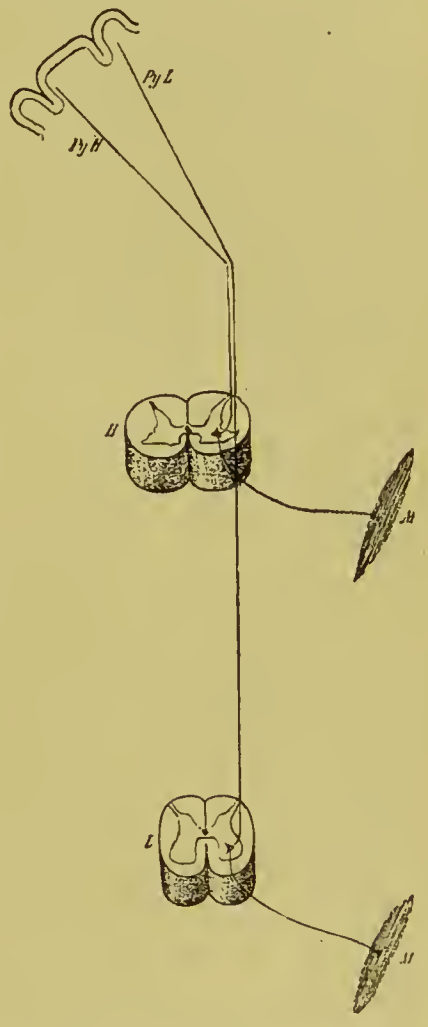


FIG. 168.—Schema of the innervation of the muscles (partly from Edinger). The radiation of the *Py*-tracts varies at different portions of the cortex (see p. 417).

(*c*) **Primary Myopathic Atrophy.**—This is a disease of the muscle, the nervous system being intact. It manifests itself by the fact that in this disease the muscle gives less response, corresponding to a simple diminution in its volume; or, if it becomes completely shrunken, there is complete paralysis; and further, by the fact that the electrical examination, as a rule, does not exhibit any trace of the reaction of degeneration. This kind of atrophic paralysis occurs in two quite dissimilar forms:

1. In *muscular dystrophia* (Erb), the myopathic form of progressive muscular atrophy (here often combined with hypertrophy or pseudo-hypertrophy).[2]

2. In *severe chronic diseases of the joints.*

The parallelism between atrophy and paralysis mentioned above is, moreover, generally present also in degenerative-atrophic paralyses, provided they develop gradually; that is, in subacute and chronic cases. A distinct disunion of atrophy and paralysis occurs only in

[1] See under Tonus.

[2] See next page.

acute degenerative-atrophic paralysis (poliomyelitis acuta, injury, etc. of the nerve, acute degenerative neuritis); here the paralysis develops more or less rapidly, but atrophy only becomes manifest in the course of weeks.

Charcot has recently discovered, in certain hystero-traumatic paralyses, a *functional paralysis* with more marked atrophy, but without the reaction of degeneration.[1] But the atrophy here is not so decided as degenerative atrophy, being rather between this and the atrophy of inactivity.

In very exceptional cases, when there is disease of the cerebrum, particularly of its cortex, there has been found a considerable muscular atrophy, which appears early, sometimes even before the occurrence of paralysis, without the reaction of degeneration. In individual cases of this character contractures were completely wanting and tendon-reflex was not increased.

Genuine *hypertrophy of muscles* occurs in *Thomsen's disease* [general myopathic spasm]; also sometimes in individual muscles, especially the gastrocnemius muscle in dystrophia musculorum; here also belongs the muscular hypertrophy which develops in the sound leg when one is paralyzed (as in long-standing infantile paralysis). Genuine hypertrophy is recognized by the increased volume, great hardness, and especially by the increased vigor of the muscle.

Pseudo-hypertrophy, on the other hand, shows increased volume, but diminished power. This occurs in dystrophia musculorum much oftener than genuine hypertrophy, but it may be developed from the latter.

Tonus of Paralyzed Muscles (*Active Spasm; Rigidity of Muscles*).—An increased tonus of the muscles that are paralyzed (rigidity, active spasm) is a characteristic, though sometimes absent, sign of those paralyses which are caused by diseases in the central neuron. This tonus may be so slight that the examiner will only notice it as a slightly increased resistance during passive motion. But it may also be so strong that even when perfectly at rest a muscle is as hard as a board, and that motion of a joint in which the muscle would be extended (that is, in which the muscle would act as an antagonizer) is entirely impossible. Thus spasm of the quadriceps prevents bending of the knee, not only passive, but also active bending, which, probably, if the flexing muscles were intact or were only paretic, would take place (*spastic pseudo-paralysis*). Patients also, even in slight degrees of rigidity, experience great difficulty in making active motions. That these spasms are not due to permanent anatomical changes in the muscles, only to muscular contraction, is proved by the fact that they are sometimes subject to striking change. If the paralyzed muscles are spastic to a high degree, often for a long time there does not develop any atrophy of inactivity.

Paralyses due to *affections of the cortex of the brain* usually manifest themselves by very early spasms. In hysteria also very decidedly active spasms occur.

Regarding *increased tendon reflex* as an attendant phenomenon of spasms, see page 359.

[1] Hereafter the abbreviation R. D. will frequently be used for "reaction of degeneration."

Atonic Paralysis.—This is characterized by diminution or loss of muscular tonus, in consequence of which there is abnormal passive mobility of the joints. This laxness is present in recent paralyses, in which the atrophic, acutely degenerative condition has not yet developed ("atonic atrophic paralysis"). It is also found in cases of chronic and long-standing degenerative paralysis (see also under Contractures). Cerebral paralyses, as hemiplegia, in rare cases may also manifest decided atony. In tabes[1] there is a tolerably marked laxness of the muscle, without paralysis.

Contractures.—In long-continued paralyses, both degenerative and simple, there develops in the paralyzed limbs a constant *anatomical shortening* of individual muscles, and, indeed, just the muscles that are chiefly spastic often shorten in spastic paralysis, but not always. On the other hand, in degenerative paralysis it is more the antagonizers of the paralyzed muscles or those of the paralyzed muscles that are strongest. Thus from the moment of paralysis the prevailing position, the posture of the affected limb, gives the first indication of the development of contracture. These contractures do not change. The motions of the limb that oppose the contracture and the stretching of the affected muscles caused by this motion are very painful.

3. The Reflexes.

1. Skin Reflexes.—By this we understand the quickly passing contractions of the muscles which are caused by an irritation applied to the skin. The stimulation of the skin usually recommended is tickling or stroking it with the blunt end of a pencil or the handle of the percussion hammer. It is well from the beginning to aim at a certain symmetry in the methods we employ; only in certain cases, especially if there is diminution of the reflex, we may endeavor to call it forth by pricking with a needle or touching it with a piece of ice, etc. The skin reflexes about to be mentioned in detail are, even in health, very different in different individuals (the cremaster reflex relatively varies least), but upon both halves of the body they are always alike. Therefore, where there are unilateral anomalies of it the most certain results of trial of the skin reflex are obtained by a comparison with the sound side. If we have like results upon both sides of the body, then it has only a doubtful diagnostic value.

We are not to confound with skin reflexes those motions that are voluntarily made. With some practice they are readily distinguished.

In the face and the upper extremities the skin reflexes are of no importance; on the other hand, the three reflexes upon the legs and abdomen are of especial diagnostic significance:

(*a*) *The reflex of the sole of the foot.*—This is produced by irritating the skin of the sole of the foot, and in health consists either in a dorsal flexion of the toes or of the whole foot, or even in motion of the hip-joint and knee. Pathologically, the reflex may be absent (weakened on one side and increased upon the other). It may be increased with reference to the amount of the contraction, with reference to its extent, as in simultaneous contraction of the other leg,

[1] See also p. 461.

motion of the pelvis or of the whole body for instance, as shorter opisthotonos; or it may occur *slowly*, or only after repeated and continued application or summation of a strong irritation. It would be influenced in its form by the tonus of the muscles of the legs: in spasm of the extensor, for instance, often, instead of a single motion of flexion there occurs repeated trembling.

(*b*) *The cremaster reflex* in men consists of a prompt upward motion of the testicle from the contraction of the cremaster which follows irritation upon the inner surface of the thigh. It is not to be confounded with the indolent contraction of the tunica dartos of the scrotum, which follows somewhat later. Sometimes the cremaster reflex is extended to the muscles of the abdomen, causing the backward drawing-in of the abdomen.

(*c*) *Abdominal reflexes.*—If the hand is quickly passed over the skin of the abdomen, there follows a contraction of the transverse abdominal muscles on the irritated side, or even on both sides. If the reflex is strong, the retraction of the abdomen is unmistakable. When the reflex is weak we sometimes only notice a slight displacement of the navel toward the side irritated. We distinguish three abdominal reflexes on each side above, within, and below the navel depression.

Fig. 169 explains *the mechanism of the skin reflexes:* the sensible irritation proceeding from the skin is conveyed by the motor fibers to the anterior horn; but the anterior horn itself is influenced by the reflex retarding fibers which pass in the pyramidal tract. It is clear that the skin reflex must be *lost* by an interruption of the reflex arc at any point, or by unsusceptibility of the skin, or by myopathic paralysis—that it must be increased with any *increased* excitability of the anterior horn or removal of the restraining reflex from the brain; also in hyperesthesia of the skin. Recently an increase of the abdominal reflex upon one side has been observed in intercostal neuralgia (Seeligmüller).

We have not mentioned a number of other skin reflexes, since they are not important. For *pupillary reflex*, the reflex closure of the lids, see under Examination of the Eye.

Of *the reflexes of the mucous membrane*, the choking reflex when the mucous membrane of the pharynx is tickled has diagnostic significance: its absence is a frequent occurrence in hysteria (anesthesia of the mucous membrane), also in bulbar paralysis (nuclear paralysis).

Of very much greater diagnostic importance are the—

2. Tendon Reflexes (*Periosteal, Fascial, Reflex*).—These reflexes are likewise short contractions. They are produced by taps upon the tendons of muscles, upon the bones and fascia, also by sudden tension of a tendon by a quick passive movement, in which, however, the muscle itself is also stretched. Both the short movement of the limb and the momentary hardening of the muscle may be made an object of examination. In order to develop the tendon reflex it is necessary to have the limb perfectly relaxed, and it is well also to divert the attention of the patient.

Whenever it is possible a comparison is to be made between the right and left limbs, but even where this cannot be done, as when the disturbance is bilateral or the two sides are disturbed in a similar

way, the greatest importance can be attached to the result of the test, because here the individual variations are not prominent, as they are in the reflexes of the skin; hence the tendon reflexes are much more important aids in diagnosis than the skin reflexes.

Tendon and *skin reflexes* may be *confounded*. In a doubtful case this can be avoided by comparing irritation of the skin alone at the given points by means of pinching, pricking a fold of skin, or by direct

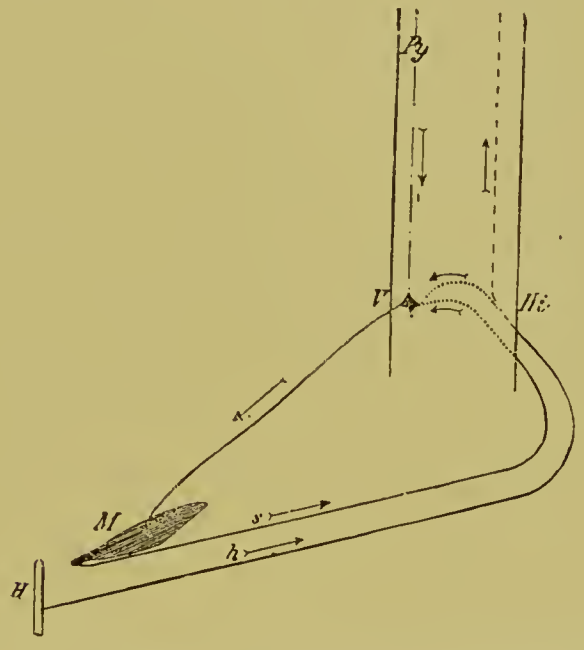


FIG. 169.—Diagram of the course of the cutaneous and tendon reflexes.

H, skin; *M*, muscle; *V*, anterior horn; *Hi*, posterior horn; *s*, the tract of the tendon reflexes; *h*, the tract of the cutaneous reflexes.

mechanical muscular irritation;[1] lastly, as in the skin reflexes, by having the patient take part in the examination by making voluntary contractions: these take place later, and hence can only deceive the inexperienced. We may be very easily misled into supposing that there is an absence of tendon reflex if the muscles under examination are not perfectly relaxed.

We enumerate the tendon reflexes according to their importance:

(*a*) **Patellar reflex** (Erb; knee-phenomenon, Westphal) consists in a contraction of the quadriceps. It is caused by striking with a percussion hammer, with the tips of the semi-flexed fingers, or with the rim of the ear-plate of a stethoscope, upon the patellar tendon. Often we must carefully seek the most susceptible point.

Sometimes we may first make the test with the leg covered, but if the result is in any way doubtful, then the knee must be uncovered. Whenever a very exact examination is to be made, the latter must always be done. In order to get the muscles completely relaxed, we must select certain positions: a favorable position is to have the limb extended at rest, with the feet resting upon the floor; another position is with the leg crossed over the other in the sitting position; a third is to have the patient sit upon a table with the legs hanging down;

[1] See below, Biceps-tendon Reflex.

with the patient in bed we pass the hand under [the thigh just above] the knee and gently lift it. As a means of inducing patients to relax the limb they are to be diverted by conversation, or they may be directed to close the fist as tightly as possible, or sometimes we may have them grasp the left hand of the examiner or press the hand of some one else.

Not only *active contraction*, but possibly also *increased tonus* of the quadriceps, disturbs the exhibition of the reflex. Even a pathologically increased patellar reflex may thus be hindered by spasm, which must be carefully guarded against. Hence, so far as possible, we must prevent any active spasm by the position (particularly by a cautious passive motion) of the knee-joint. It may also be interfered with by deformity and stiffness of the joint.

With very rare exceptions the patellar tendon reflex is always present in health, and both sides are equally strong.

The author cannot forbear saying that he regards as impracticable the designation "Westphal's sign" for the *absence* of patellar reflex—notwithstanding his very high regard for this meritorious investigator, who is deserving of the honor—because this designation could easily be confounded with the opposite (as, that Westphal's sign meant patellar reflex).

(*b*) **Tendo-Achillis Reflex and Foot-phenomenon.**—Striking upon the tendo Achillis, and often only on a very limited portion of it, in health generally causes a reflex contraction of the gastrocnemius (and soleus) with slight plantar flexion of the foot. In doing it, it is best to lift the foot by taking the malleoli with the left hand (the foot of course being bare).

By *foot-phenomenon* we designate the contraction of the same muscles if there is a continuous contraction, a passive dorsal flexion of the foot, often best excited by a quick passive motion (stretching the tendons, also the muscles); a reaction then takes place in a series of rhythmical contractions of the plantar flexors or a long series of contractions—foot clonus, foot-phenomena, dorsal clonus. This latter phenomenon is not really a pure tendon-reflex; rather in part it is dependent upon direct irritation of the muscles as a result of stretching. But it has exactly the same diagnostic significance as increased tendon-reflex, for it does not at all occur in health, or, at most, only temporarily, as when one is very tired.

(*c*) **Tendon Reflex of the Upper Extremities.**—Here they do not have the same diagnostic importance [as those under (*a*) and (*b*)], particularly because they are very often absent in health. Striking the flexor tendons at the wrist-joint, the biceps at the bend of the elbow, the triceps tendon close above the olecranon, generally causes a slight reflex contraction; in the two latter we must be careful not to strike the muscle itself.[1]

(*d*) **Periosteal and facial reflexes** are elicited by striking the latter and the bones—the tibia: patellar reflex; bones at the wrist-joint: biceps, even pectoralis reflex. We not infrequently observe them in health, but very particularly when there is increased tendon reflex. Not wholly unimportant also are the bone reflexes which are manifest

[1] See Mechanical Irritation.

in the muscles of the face from blows upon the chin and upon the nose; they are absent in bulbar paralysis, and are present in paralysis of the facial tract above the bulb.

The mechanism of the tendon reflex is made clear by Fig. 169, page 459. We see that for its production it is necessary to preserve the integrity of the reflex arc: (*a*) tendons; (*b*) sensitive (that is, centripetal) nerve; (*c*) posterior root; (*d*) anterior horn; (*e*) motor nerve; lastly (*f*) muscle. But we take note of the influence upon these of restraining fibres in the pyramidal tract, which may be cut off, and also may possibly be temporarily irritated. Interruption of the pyramidal tract, which is manifest by its secondary degeneration as far as the anterior horn, or cutting off of the pyramidal tract by primary degeneration, causes increase, therefore, of tendon reflex, as in *cerebral paralyses*, spinal paralyses from disease of the pyramidal tract, in *myelitis transversa*, *amyotrophic lateral sclerosis*, *spastic spinal paralysis;* but also increased irritability of the spinal cord itself, as in *strychnia-poisoning*, *tetanus*, *lyssa*, *neuroses*, and particularly sometimes in *hysteria*. On the other hand, the *tendon reflexes are diminished or* are *lost* in disease of the anterior horns, of the peripheral nerves, of the posterior roots or their connection with the anterior horns (*poliomyelitis*, *spinal progressive muscular atrophy;* any *disease of the peripheral nerves; tabes dorsalis*—here diagnostically very important; *myelitis*, *tumors*, *hemorrhages*, if in certain locations—that is, if they disturb the gray substance for the arm or leg).

It follows from what precedes that the increase, and also in many respects the diminution, of the tendon reflexes goes parallel with increased or diminished tonus of the muscles. And, in fact, tonus seems to be genetically related to tendon reflexes. In this sense it is also of interest that the predominant reflexes of the arm are the flexors, of the leg the extensor of the knee, the plantar flexor tendo Achillis, reflex of the foot, and that exactly corresponding with a recent spastic paralysis of the arm we are apt to have flexor spasm of the arm and extensor spasm with paralysis of the leg at the knee and ankle.

Westphal's view that the "tendon reflexes" are not reflexes, but that they are always, when elicited by the prescribed methods of testing, due to the direct irritation of the muscles by stretching and concussion, is to be regarded, especially as respects patellar reflex, as definitely refuted. Nevertheless, we must still agree that the ordinary method of examination for the foot-phenomenon in this respect is not free from objection (as has been urged by others also, as by Jendrassik): the brusque dorsal flexion of the foot must necessarily stretch the gastrocnemius—here it may be due to the effect of stretching of the muscle added to that of the tendon.

Mixture of tendon reflex and direct muscular irritation from stretching the muscle probably also occurs in executing "brusque passive motion" of the limb (very quickly bending it and extending the knee-joint, etc.), which is very strongly to be recommended for determining a slight degree of increased tonus of the muscles.

4. Electrical Examination of the Nerves and Muscles.[1]

REGARDING THE PHYSICS, AND THE INSTRUMENTS EMPLOYED.

The electrical examination of the motor nerves and of the muscles consists in an electrical irritation of these organs at points where they are situated subjacent to the skin.

For the electrical examination we employ the *faradic induction current* of the secondary spiral of a Dubois-Reymond sliding apparatus and the *constant current* of a galvanic battery. We cannot give a full description of these instruments here. They are fully described in works upon Physics. We speak only of what is of particular importance for electrical diagnosis.

It is especially necessary that the [faradic] apparatus should be so made as to enable the user to graduate *the strength of the current* in any way that may be required. In the faradic battery this is done by changing the position of the outer or secondary coil with reference to the inner or primary one: the greater the distance between the coils the weaker the secondary current becomes, assuming always that the strength of the primary current, usually supplied by a chromic acid element, remains constant. If both coils are pushed completely one over the other the greatest possible strength of the secondary current is obtained. In this position we say that the distance of the spirals is equal to 0. The further the outer spiral is removed from this point the weaker the secondary current becomes. A scale is fixed to the apparatus for a measure of the degree of weakening or, in other words, for the strength of the secondary current. This scale indicates in centimeters and millimeters the displacement of the outer from the inner coil. If the coils are entirely one over the other the outer one indicates 0; if it is drawn out one centimeter we speak of one centimeter spiral distance, etc. The more centimeters of spiral distance the weaker the current. Now it is clear that this designation of the strength of the secondary current according to the spiral distance is not absolute, as the strength of the secondary current depends upon that of the primary. The *strength of the galvanic current* is regulated in a rougher manner by varying the number of elements. For finer alterations of the strength of the current a special apparatus called a rheostat is used, the handling of which is very simple. [The galvanic batteries now made in the United States and England usually have a rheostat as a part of the outfit. It is much better to use it, for two reasons: all the cells of the battery are drawn from alike, since all can be thrown into the current at the beginning of each sitting; the gradations in the strength of the current are made without shock to the patient.] The strength of a current can be determined by a so-called absolute galvanometer, of which we will speak later on.

The current is conveyed to the body by an electrode previously moistened with warm [preferably salt] water. In making the examination, one of these is always the *indifferent* one—that is to say, it merely serves to close the current that is flowing through the body; the other is the "*differentiating*" or examining one. For the selection

[1] Of course it is not necessary here to go into particulars. Hence we refer the reader to special works, particularly to Erb's classical work, *Electro-therapy*.

of the size of these electrodes there are two very different determinative points of view.

The one takes into account *the resistances of the skin.* This is by far the strongest of the resistances which the tissues of the body, with the exception of solid parts of bones, offer to the current. But since the resistance diminishes in proportion to the square of the cross-section it is best to make both electrodes as large as possible in order to lose the least possible strength of current in passing through the skin.

The other point of view is to some extent opposed to the first. As has been said above, since we have in view an irritation through the skin of nerves and muscles superficially situated, we must be careful to always have the greatest possible portion of the strength of current which the apparatus supplies expended for the irritation of the respective nerves or muscles. A sharp distinction must be made between "total-current-strength" and that portion of it which is employed for the irritation, "irritation-current-strength." In order that the latter may constitute as great a proportion of the former as possible, it is necessary in the passage through the skin to concentrate the current upon a conductor whose cross-section approximates as closely as possible that of the subcutaneous organ; hence for the purpose of irritating a nerve the cross-section upon the skin must be very small; therefore for the examining electrode we select one as small as possible. But this fact, already mentioned, must be remembered, that the "total-current-strength" is considerably diminished by the diminution of the cross-section of the current, just in the skin which in itself offers a strong resistance, and this is true with the galvanic current in a higher degree than with the faradic. For this reason, and especially on account of the disagreeable secondary effects of too dense a current (pain, cauterization of the skin), it is necessary in making examinations to select electrodes whose cross-section is somewhat larger than that of the nerves.

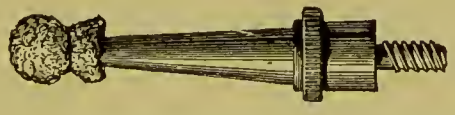


FIG. 170.—"Fine" electrode of Erb (natural size).

From this explanation it is evident that the "indifferent" electrode may be of indefinite size, limited only by considerations of convenience. The "examining" electrode, however, must be small, and in examinations of the nerves and some muscles, if the faradic current is employed, it may be very small. We recommend the fine sponge electrode of Erb, represented in Fig. 170.

The galvanic current, on the other hand, does not permit the use of such small electrodes, because it is more sensitive to resistance and besides, by its greater density, it damages the skin. For this reason, in examining with the galvanic current, we employ a somewhat larger electrode. Now since we can always measure the total-current-strength of the galvanic current by means of a galvanometer,[1] the attempt has been made to arrange conditions, which, upon the basis

[1] See below.

of the "total-current-strength," furnish as exact conceptions as possible of the "irritation-current-strength," which is the only one for ultimate consideration. As everywhere else, so also in the passage through the skin, the density of the current is in inverse proportion to its cross-section. If the "total-current-strength" and the cross-section of the current are known, the density of the current—that is, the strength of the current in terms of the unit of measure—may be best calculated pro square centimeter of the cross-section. Given an examining electrode of a definite known cross-section, we may determine the density of the current in the passage through the skin in the manner indicated and thus form an at least approximate idea of the density and the strength of the current which is present in organs whose cross-section is approximately known and which lie directly under the skin (nerves, muscles). Examining electrodes of definite cross-section [as standards] have been constructed for the constant current for the purpose of getting as fair a relation between the "total-current-strength," the "total-current-cross-section," and the "irritation-current-strength," although this relation is only imperfectly known. Unfortunately there are several of these: but we take into consideration only the following: that of Erb, of 10 square centimeters cross-section (either square, 3.3 cm. on a side, or circular with a diameter of 3.5 cm.; and that of Stinzing, round and somewhat convex, 3 square cm. in cross-section and 2 cm. in diameter. With every record of an examination there should always be a statement of the size of the electrode employed.

As yet we have no simple, practicable method for measuring the "total-current-strength" in making examinations with the faradic current, since the apparatus hitherto constructed for that purpose (faradimeter) is too complicated and expensive. Under these circumstances nothing remains but to forego absolute dosage in making the faradic examination. The distance of the coils may be noted, which may signify a different strength of current according to the construction of the respective sliding apparatus and the strength of the elements which furnish the primary current, but which, nevertheless, has a certain comparable value for the different examinations made with the same apparatus. Moreover, a certain exactness may be attained if, from time to time, the user tests the effectiveness of his own sliding apparatus at a given distance of the coils upon a healthy person, best upon himself, for instance on the left ulnar nerve at the wrist, and if he observes the points discussed later on. Besides it is important to be certain in another direction: although the faradic current is less influenced by the resistance of the skin than by the galvanic, nevertheless an exceptionally great, or exceptionally small, resistance of the skin at any given place of examination may so very much diminish, or relatively so much increase the strength of the current that the examiner considers that the irritability, for instance of a nerve, is considerably smaller, or greater, as the case may be, at such a place than it really is. In order to counteract this error we may determine the resistance to conduction for all points of the skin at which faradic irritation has been made by ascertaining the declination of the needle of a galvanometer when a galvanic battery with always the same number of elements

has been inserted.[1] The judgment thus obtained in regard to the resistance of a spot of skin to conduction of the constant current may be transferred to the faradic current. It must be remembered, however, that only considerable deviations from the normal come into consideration for this latter current.

The galvanic current, however, we measure directly, according to an absolute measure, in *milliampères*, abbreviated, 1 M.-A. $= \frac{1 \text{ volt}}{1000 \text{ ohms}}$ (see text-books upon Physics). To ascertain the number of milliampères used we employ a so-called absolute galvanometer. The total strength of current indicated by the galvanometer is then divided by the transverse section of the examining electrode in such a way that, for example, with a total strength of 2.5 M.-A. and an electrode of 12 sq. cm. transverse-section to a sq. cm. of the skin, a current of $\frac{2.5}{12}$ M.-A. is given off; now, after what has been already said, this fraction is not an exact measure of the extent of irritation to which the nerve is subjected as it lies buried beneath the skin, and generally also in a layer of fat of varying thickness; but at any rate, it gives a result which is of value for comparison if applied in all examinations. If we employ a normal electrode, then we can note: Norm. electrode Erb (10 sq. cm.) 2.5 M.-A., or $\frac{2.5}{10}$ M.-A. (N. el. Erb).

This comparison of the total strength of the current with the absolute measure is now-a-days indispensable: it has, it is true, only a value which is, in a certain sense, circumscribed. A difficulty which at present is tolerably successfully overcome consists in the fact that the conducting resistance of the skin for various reasons declines as the current passes through it, and therefore, although only in a slight degree, the strength of the current increases so long as the electrodes rest upon the body, and hence, also, from the moment when the galvanometer is switched in to the instant when the needle comes to rest. With the new galvanometers (especially Edelmann's horizontal galvanometer, but also with the instruments of Böttcher-Stöhrer and Hirschmann), by an appropriate check, this space of time is satisfactorily shortened. Stintzing is to be credited with very exact examinations regarding these points.

A much more considerable difficulty, which has already been hinted at several times, and one which probably will never be satisfactorily solved, consists in the fact that we cannot concentrate our current upon the nerve or muscle to be examined, because, covered by the skin and partly also by subcutaneous fat and fascia, they lie in a medium which itself is a good conductor, which diverts to itself a part of the current. Furthermore, we cannot, even with the least accuracy, calculate how great is the portion of current which ultimately reaches the nerve or muscle; for the situation of these structures with reference to the skin and their immediate surroundings is extremely variable at different parts of the body, and also in different individuals. On this account not only is the irritability of muscles and nerves as measured by the

[1] See below.

"total current-strength" influenced in an uncontrollable manner, but also, according to Erb's investigations, even the quality of the contractions are affected.

There follows from the foregoing, first of all, the practical point that, in spite of our ability to measure the strength of the total current, we are taught to bear in mind the individual peculiarities of the nerves (muscles) to be examined, in their relation to the skin, in interpreting the results of the examination, so as to supply, as far as possible, the want of exactness in our calculation; and it follows, further, that it is superfluous, and even a source of error (because it withdraws our attention from the more important points of view), if we strive after exactness in electrical examination by the fineness of the apparatus, especially of the galvanometer—an exactness which, let it be said once for all, the examination cannot have. Of what use is it exactly to determine the strength of the total current to within one-tenth of a M.-A., if the anatomical conditions of the nerve which is to be irritated cause an inexactness impossible to calculate amounting to whole milliampères?

How to Distinguish the Poles Quickly.—In the faradic current the poles come but little into consideration—namely, only so far as to know that the cathode (the negative pole) of the opening current of the secondary coil has a stronger irritating effect than the anode (the positive pole). In the galvanic current the poles are widely different. They are always marked on the apparatus by the signs of plus and minus, but as they may be reversed by screwing on the conducting wires in an opposite way, it is necessary to always test them anew. The simplest way is to employ a very mild current, and then to place the two electrodes upon the cheeks; upon the side of the anode we experience a peculiar indefinable taste upon the tongue and the mucous membrane of the cheek of that side, while on the cathode side there is no sensation; or we place the wires of both poles about 1 cm. apart upon a piece of wet blue litmus-paper: the anode colors it red.

By a current-changer we are able to reverse the poles—that is, to quickly make the anode the cathode, and *vice versâ*.

METHODS OF EXAMINATION IN GENERAL, AND THEIR PHYSIOLOGICAL RESULTS UPON THE LIVING HUMAN BODY.

As a foundation to what is here to be spoken of, we refer most urgently to the text-books upon physiology or upon electro-therapeutics, especially to what is taught regarding electrotonus and the laws of contraction (Pflüger). Unfortunately, we cannot enter upon these subjects here. [The student is referred to Landois and Stirling's *Physiology* for an excellent presentation of Electrotonus—law of contraction.]

The electrical examination consists in the production of muscular contractions by means of both kinds of current, and sometimes by irritation of the muscles themselves (*direct irritation*), and by irritation of nerves (*indirect irritation*). The latter is generally made before the former, and thus we have to make use of an *indirect faradic* and *galvanic* and a *direct faradic* and *galvanic* examination. As previously stated, the extent of the irritation is always a matter of uncertainty to us

(distance of the coils ; total strength of the galvanic current in M.-A. is known). We draw our conclusions from the results of the examination :

(*a*) From *the degree of excitability* of the nerve (muscle) by determining with what strength of current there follows the first, smallest, just noticeable, or minimal contraction ; sometimes also by determining the extent of irritation which is necessary with the galvanic examination to cause a tetanic contraction. The minimal contraction is observed at the muscle or by the movement of the joint. The comprehension of these minimal contractions (still more of galvanic tetanus[1]) by the individual examiner is, to a certain extent, variable, and a source of inexactness.

(*b*) With reference to *the quality of the reaction* in the direct irritation of the muscle with the galvanic current; that is, the character of its contractions and its "law of contraction," regarding which we will speak more at length below.

Since the electrical currents, except they be very strong, only stimulate by sudden oscillations in the current, the faradic current, because it consists of a great number of opposing currents of short duration, causes a tetanic contraction proceeding from the nerve as well as from the muscle itself, which continues while the electrode remains with the current closed; the galvanic current, on the other hand, indirect as well as direct, produces its effect only at the instant of its entrance. In both instances, if the current is sufficiently strong, contractions take place—the closure contraction at the instant of closing, the opening contraction at the instant of opening the current, and at the instant of its exit. But while with the nerve exposed (Pflüger) at the cathode [represented hereafter by Ca] (negative pole), only the closing of the current, and at the anode (positive pole) [represented hereafter by An], only the opening of the current occasions a contraction, we find that with the nerves and muscles of the living man there is another law of contraction, which at first sight seems to contradict Pflüger's law, but the contradiction is easily explained if we consider the peculiar conditions of the experiment which are present in the living human body; that is, in a nerve which is not exposed. Regarding this, as it were, "clinical law of contraction," we must now go a little more into detail :

General Methods and Explanation of the Terms Employed in Galvanic Examinations.—The indifferent electrode is placed upon the sternum, the examining electrode (normal electrode) upon the nerve or muscle to be examined. With the current-changer we close the current so that the examining electrode is the cathode—that is, we make the "cathodal closure" CaS [S = *Schliesung*, closure]; there results a contraction, C, thus it is CaSC; then we open the current, thus making a cathodal opening, CaO : sometimes there is CaOC ; then with the current-changer we reverse and close the current, so that the examining electrode becomes the anode, An, making AnS : we sometimes have AnSC, then likewise at the end AnOC. With a very strong current we have, upon CaS and with the current remaining closed, a tetanic contraction : CaSTe.

1. **Stimulation of Nerves.**—These opening and closing contractions at the cathode and anode do not all of them occur with the same

[1] See this.

strength of current. On the contrary, if we begin with a weak current and under continuous closing and opening at both poles we increase the current, we first notice that there is a slight contraction, when at a certain strength we close the current in such a way that the examining electrode becomes the cathode; that is, as soon as we make the cathodal closure, CaS. Then, while keeping the same strength, if we open the current (CaO) no contraction follows, and this is also true if we reverse and close and open at the anode (AnS, AnO). If the current is further increased we see a corresponding, more pronounced CaSC, but with a certain strength of current there also now results a contraction with AnS, and then usually also immediately with AnO: AnSC, AnOC; as yet at the cathode there is never any result. Only when the current is very strong, under which the cathode-closure-contraction changes to tetanus (CaSTe) there is a weak contraction with the cathode opening.

All these contractions, whether weak or strong, are short, lightning-like. The CaSTe is of course an exception to this. It consists of a rather short tetanic continuance of the lightning-like CaSC.

From this there results the following scheme of the laws of normal contraction with galvanic stimulation:

Nerve x:

(*a*) Weak current: no contractions at all;

(*b*) Current a little stronger: CaSC gives feeble, short contractions;
CaO negative;
AnS "
AnO "

(*c*) Strong current: CaSC gives strong, short contractions;
CaO negative;
AnS gives feeble, short contractions;
AnO " " " "

(*d*) Very strong current: CaS " tetanus of short duration;
CaO " feeble, short contractions;
AnS " stronger, " "
AnO " " " "

Or also, in brief: with a certain moderate strength of irritation there is CaSC and AnSC (AnOC is not taken into account), but CaSC is greater: CaSC>AnSC.

The contractions are, all of them, short, lightning-like.

2. Stimulation of Muscles.—In this case, as far as possible, we avoid a simultaneous irritation of motor nerves, especially the one which supplies the muscle being tested. The electrode is, therefore, placed as far as possible from mixed and motor nerves, particularly from the place of entrance into the muscle. We then find that with a moderate strength of current CaSC takes place, and that a slight increase is sufficient to cause AnSC; on the other hand, that AnOC frequently, also CaOC almost always, does not occur even with the strongest currents.

It is remarkable that the effaceable closure-contractions, especially AnSC, are not quite so short as those which are produced from the nerves. This is very striking in some individuals, less so in others.

The examination here also normally may be briefly stated as follows: with x M.-A CaSC>AnSC, with the additional statement: contractions very short, or AnSC a trace slower than CaSC.

METHOD OF EXAMINATION IN DETAIL.—NORMAL CONDITION.

Preliminary Remarks.—In examining individual nerves and muscles we must strive most earnestly to employ exactly similar methods. In the first place, in examining nerves, we should use Erb's fine electrode for the faradic current, and either Erb's or Stintzing's normal electrode for the galvanic current. With the galvanic current especially we should always make about the same pressure upon the electrode, increasing the pressure only when there is a very firm layer of fat, in order in this way to equalize to some extent the effect of the fat-layer. We are always to examine homonymous parts together; that is, the right, then the left radial, the right, then the left median; or, when the disease is unilateral, the nerve (muscle) of the sound side always first.

1. Points of Stimulation.—In what follows we give the points of stimulation of the nerves and the so-called motor points of the mus-

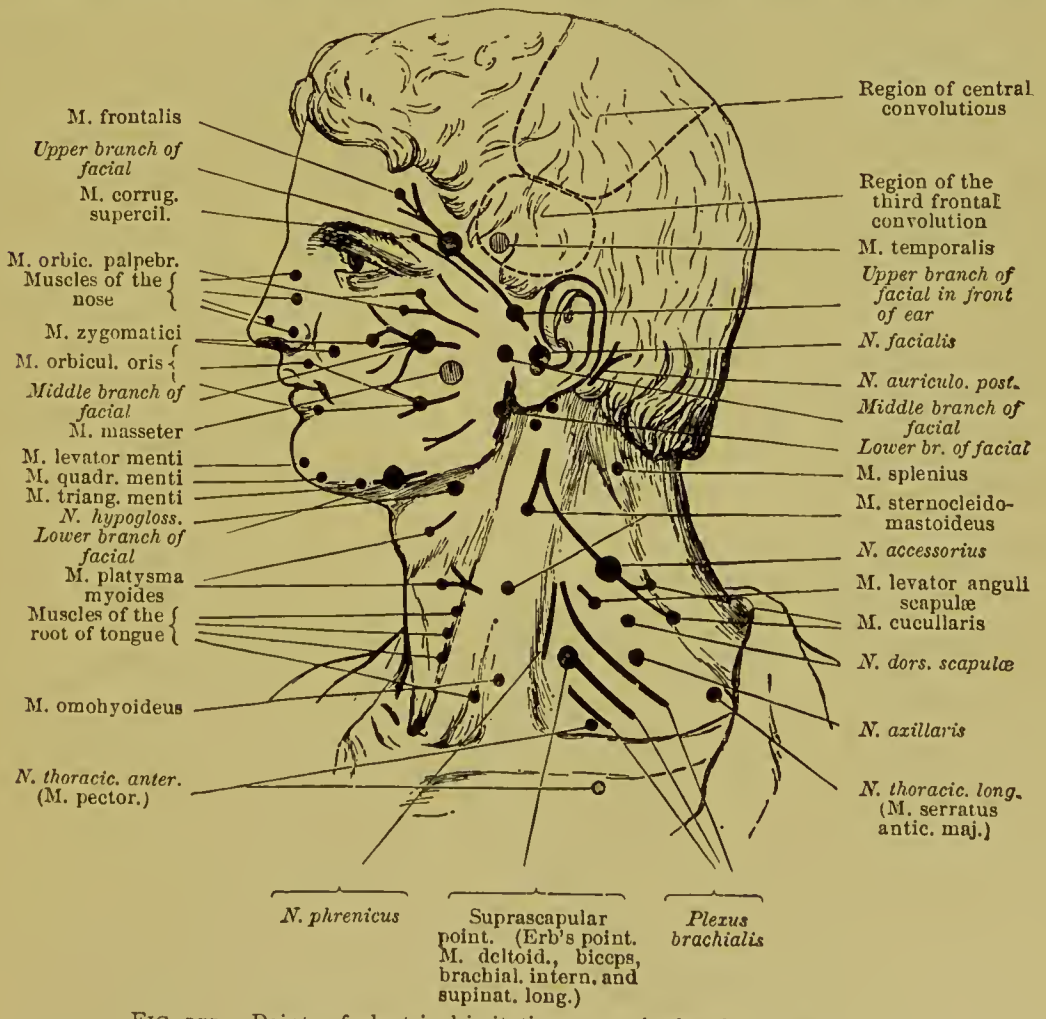


FIG. 171.—Points of electrical irritation upon the head and neck (Erb).

cles (for which we are indebted to the investigations of Duchenne, v. Ziemssen, Erb; the illustrations are taken from Erb's *Electro-thera-*

peutics. These points frequently correspond to the points where the nerves enter the muscles, and hence are essentially also the nerve-points. In examining the muscles themselves we place the electrode

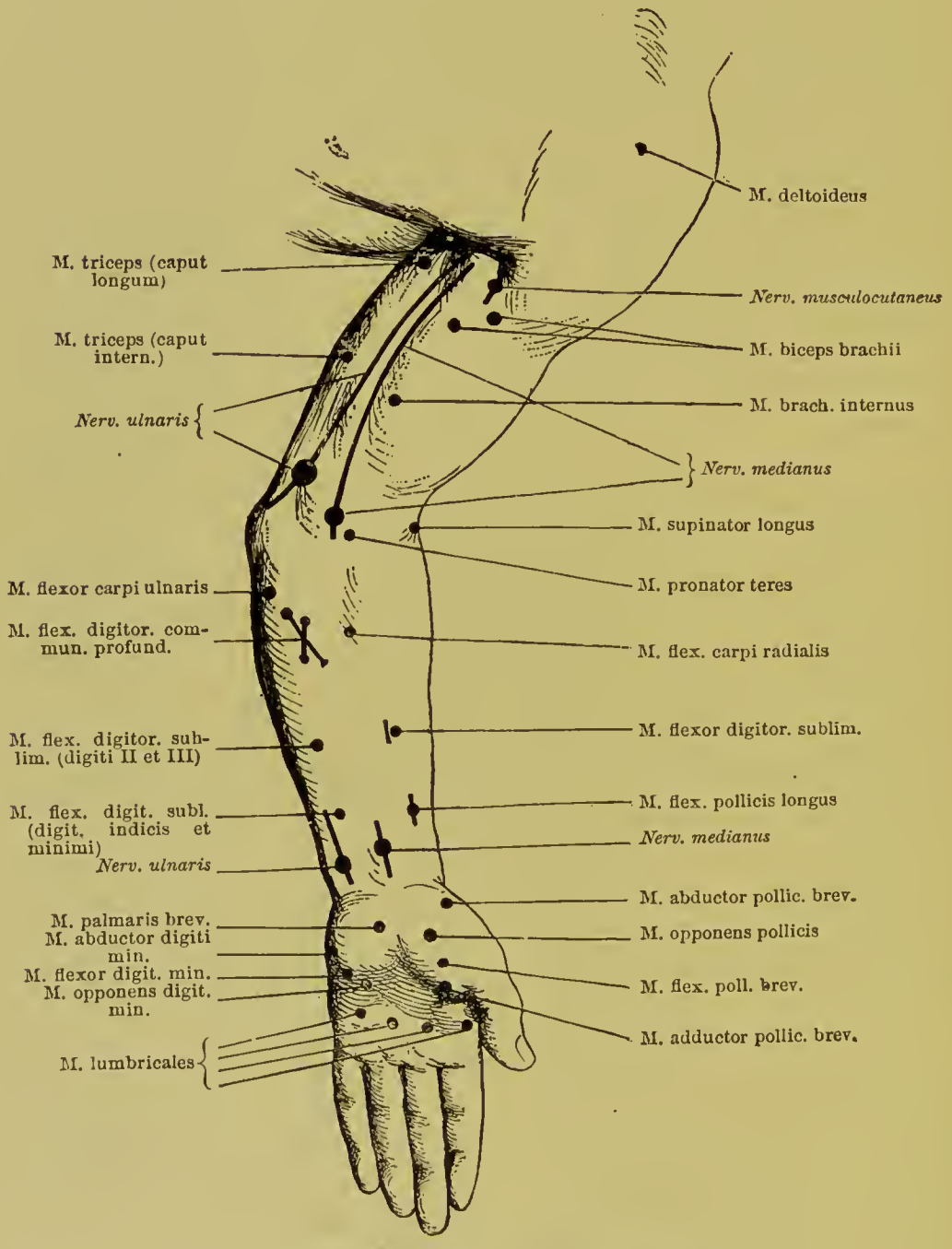


FIG. 172.—Points of electrical irritation upon the arm (Erb).

upon the fleshy part of the muscle, avoiding, as far as possible, both of these related points.

The points most distinct in the figure correspond to the chief places for applying the stimulation. In the faradic examination we seek

carefully in the course of the nerve for these most excitable points; that is, for those places where they lie nearest the skin.

Remarks regarding Fig. 171: We observe particularly the upper, middle, and lower facial (the three most distinct points upon the face). At the brachial plexus we notice Erb's point [the supraclavicular point], from which the following named muscles may be simultaneously stimulated: deltoid, biceps, brachialis anticus, and the supinator longus.

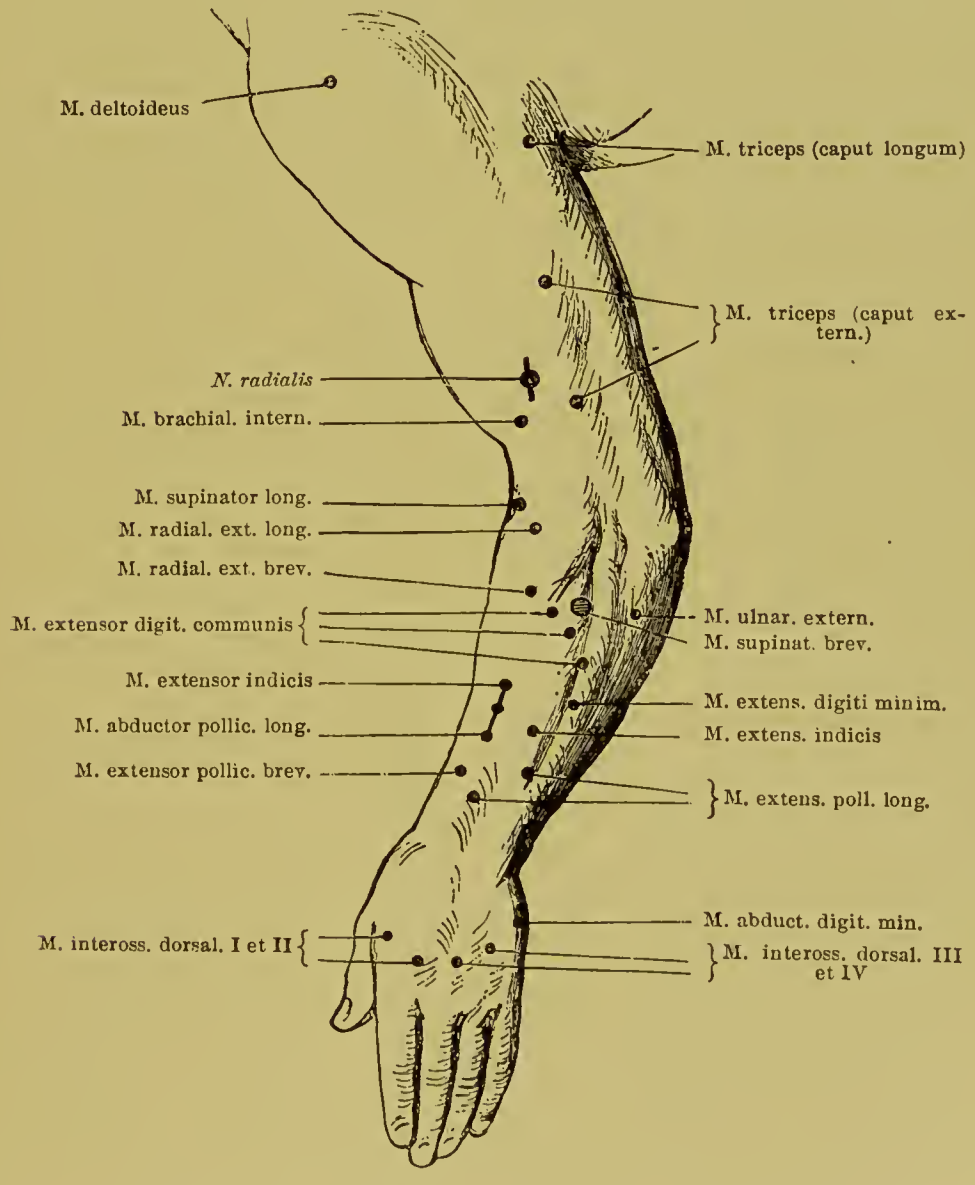


FIG. 173.—Points of electrical irritation upon the arm (Erb).

The tongue and soft palate will be best directly irritated with an electrode that is isolated as far as to the end (which may be done by simply winding it with adhesive plaster).

A strong galvanic current should never be used upon the head.

Remarks regarding Figs. 172 and 173: We examine the arm in the

position of moderate flexion and slight pronation, but the muscles are to be relaxed (hence the arm must rest comfortably).

The radial nerve lies deeply, especially if the muscles are well developed. The ulnar nerve lies in the sulcus of the internal condyle of the humerus, and can be felt with the finger here and for some distance upward.

Position of the Indifferent Electrode.—In stimulating the radial nerve and the ulnar and median at the elbow it is best to place the indifferent electrode upon the sternum; on the other hand, for the ulnar and median at the wrist-joint it is best to place it on the dorsal side of the wrist; and this also is the point most favorable for stimulation of all the muscles of the forearm and hand, because it excludes any accessory stimulation.

Remarks upon Figs. 174–176: It is very difficult to stimulate the ischiatic nerve. It can only be done by pressing the electrode in

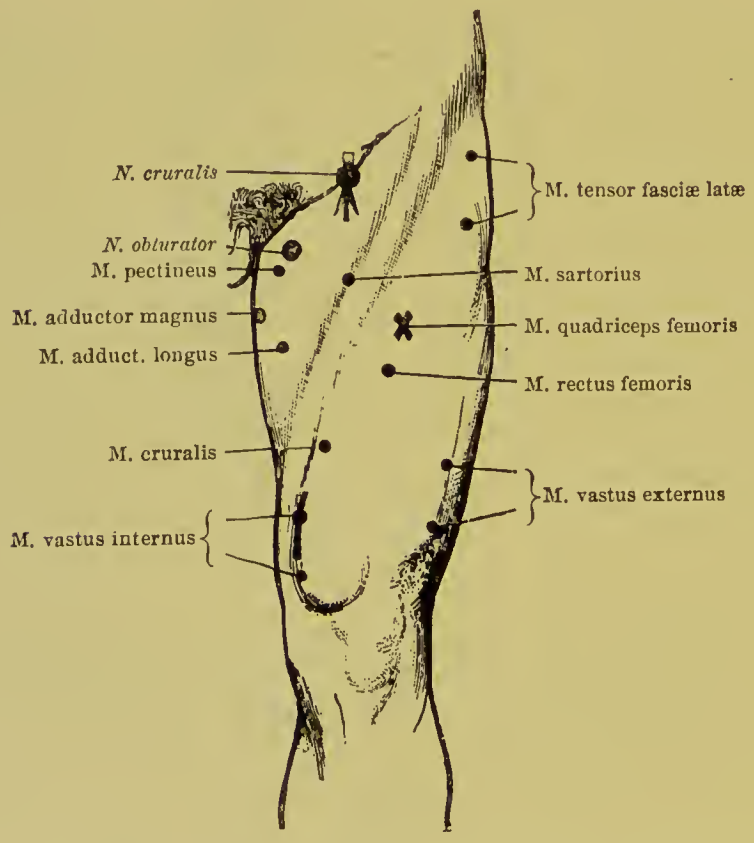


FIG. 174.—Points of electrical irritation upon the upper part of the thigh (Erb).

deeply and employing a strong current. We can easily find the peroneus nerve if we feel for the head of the fibula and go inward and upward from this.

Upon the back, since the nerves almost nowhere lie sufficiently near the surface to permit of indirect examination, we have to do almost exclusively with direct muscular irritation. It is superfluous to make more exact statements regarding the simple topographical relations.

2. Method of Conducting the Examination.—We demonstrate this upon a single nerve-muscle, and for this we take the radial. We always begin with the faradic current, and this for good reasons, which have recently been made more strong (relations of the "resistance to conduction"—Stintzing), which we cannot enter upon here.

(*a*) **Faradic Examination.**—(*a*) **Nerve.**—The indifferent electrode is placed upon the sternum, the examining electrode, that is, Erb's "fine" electrode, held as a pen in writing, is placed upon the radial nerve [the musculo-spiral] where it turns round the humerus in the middle of the arm: here tolerably deep pressure is necessary. The induction coil is now pushed out till the minimal contraction is produced; in doing this we should feel for the nerve with the electrode, which can only be learned by practice, and cause it to twitch: it is at

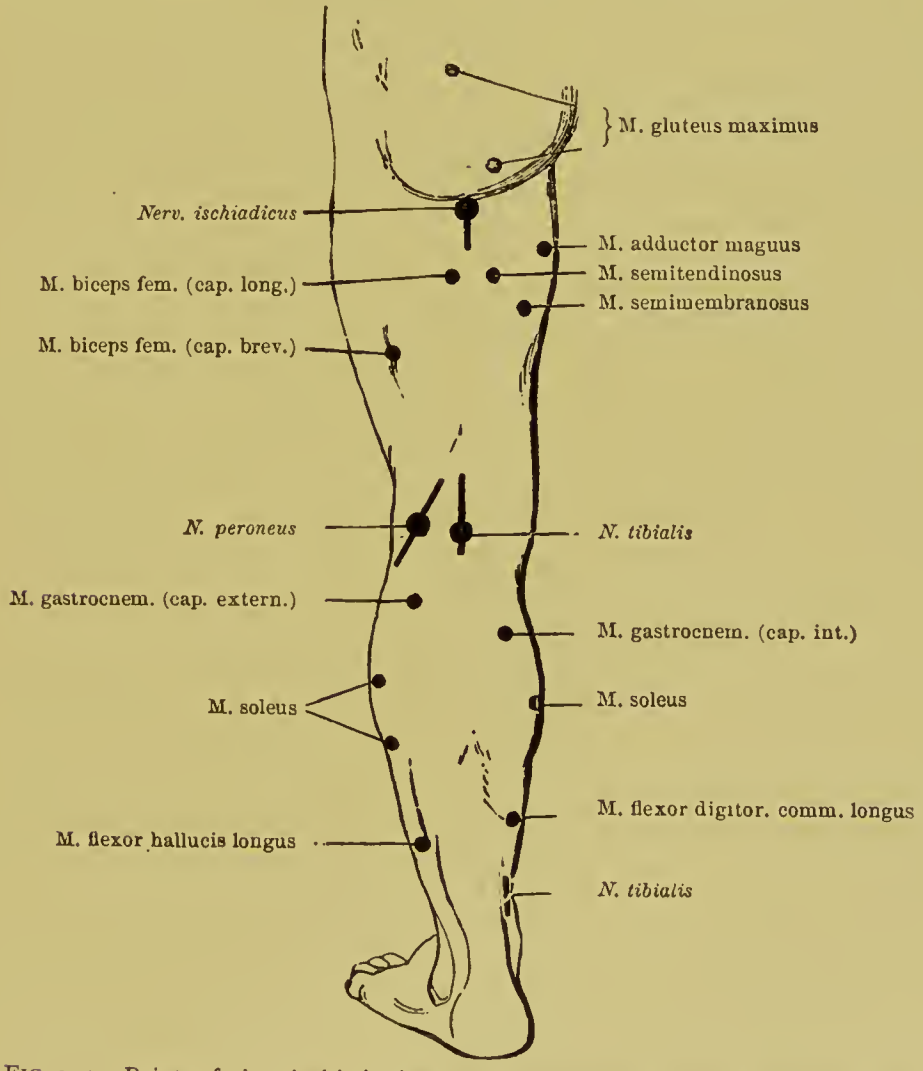


FIG. 175.—Points of electrical irritation upon the back of the lower extremity (Erb).

that instant that the minimal contractions usually take place. The distance to which the induction coil is pushed out is now read off and noted: for instance, "minimal contraction," or, abbreviated, "C, in the extensors and supinator," or "in the supinator alone with S. D. (spiral distance) of 120 mm."

Then the resistance of the skin to conduction at the points where the indifferent and examining electrodes were placed must be determined. For this purpose the fine electrode is changed for the normal electrode, which latter is put upon precisely the same place where the stimulation was applied; ten elements of the galvanic battery are put in circuit, and the declination of the needle on the absolute galvanometer is read at the instant when the two electrodes have been on for just thirty seconds with the current closed.

It is necessary, as we have before emphasized, to determine the

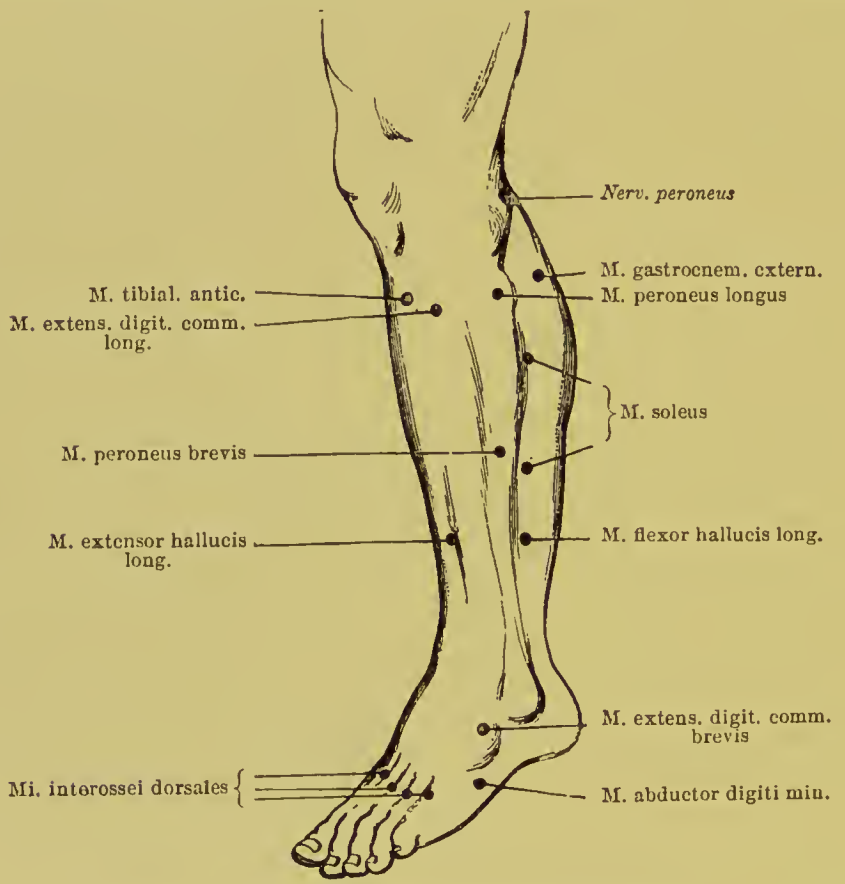


FIG. 176.—Points of electrical irritation upon the leg (Erb).

"conductive resistance" exactly in the manner described by Erb. The fluctuations in the conductive resistance, and with it (in an opposite sense) the strength of the total current, are, in fact, during the examination very slight, and can ordinarily, as has been shown most accurately by Stintzing, be neglected. But in some cases it happens that at the point of examination the skin is very tender or abnormally dense; in which case, of course, with the same separation of the coils of the same apparatus, we have relatively a stronger or relatively a weaker current, and we find a minimal contraction with a large or with only a very slight conductive resistance. This result we would refer to an increased or diminished irritability of the nerve if we had not ascertained by the galvanic determination of the "conductive resistance" that the skin was the cause of the variation. Extremely instructive examples illustrating this point are given by Erb in his *Electro-therapeutics*.

In other words: whenever we are making an electrical examination we must know what strength of total current we are employing. Since we are not able with a simple method to determine this directly with reference to the faradic current, we must endeavor to form an opinion of the total strength of the faradic current (with a certain definite separation of the coils) by bearing in mind the total strength of the galvanic current which is caused by a certain number of elements (always the same).

If we examine a number of nerves at the same time, we first determine the minimal contraction for all, and then the conductive resistance; and after we have examined the nerves we can at once make the faradic examination of the muscles.

It is always well to follow the faradic examination with the galvanic, and in this way, with a good deal of practice, we can form an opinion regarding the relation of the conductive resistance at the different points of stimulation of the nerves, and can make a counter-judgment regarding the faradic result by a comparison of the number of elements used each time and the absolute strength of current that is obtained. But then there must always be given in the record of the galvanic examination both the number of elements and the strength of the current in M.-A.

We wish that the direction given above, that the galvanometer should be read when the electrodes have been in place just thirty seconds, could be carried out in all efforts at electro-diagnosis, because otherwise the marked increase of the current at the beginning, just after the electrodes have been applied, could easily occasion great inequalities.

(*β*) **Muscles Supplied by the Radial [Musculo-spiral] Nerve.**—We use a somewhat larger electrode, stimulate the fleshy part of the individual muscles, and, lastly, determine the minimal contraction; the determination of the conductive resistance is not necessary.

Under some circumstances there comes into consideration the quality of the muscular contraction in indirect and direct faradic stimulation. (See under Reaction of Degeneration, page 478 *f.*)

(*b*) **Galvanic Examination.**—(*α*) **Nerve.**—Place the indifferent electrode upon the sternum and the examining electrode (with somewhat strong pressure) upon the radial [musculo-spiral] nerve where it passes around the humerus; close the cathode three times; if the result is still negative, increase the number of elements; again close the cathode three times, and so on until the minimal contraction is found. Then switch in the galvanometer and read off the strength of the total current. (Galvanometers that have a very good arrangement for damping the vibration of the needle can remain switched in during the examination.) Now determine the minimal AnSC in the same way (but it may be omitted). Usually we may be satisfied with this. The next point of interest would be the determination of CaSTe. (Regarding variations in the quality of the reaction, see under Reaction of Degeneration.)

(*β*) **Muscles of the Radial.**—We proceed as in the case of the nerves, but sometimes we may place the indifferent electrode upon the wrist, dorsal side.[1] It is always necessary to determine the minimal CaSC

[1] See below.

and minimal AnSC; but, before all, the most exact observance of the character of the contraction,[1] whether it is "lightning-like" or "slow," and in this direction we not only observe the minimal contraction, but also whether it is a stronger or strong contraction.

Summarized, the scheme of examination—for instance, in diminished irritability of the left radial nerve—would be as follows:

(a) Faradic examination:

(α) Nerv. radial. r. 120 S.-D.[2] (extensors and supinator): variation of needle, ten elements, 6 M.-A.
Nerv. radial. l. 87 S.-D. (extensors alone),
80 S.-D. (supinator longus): var. needle, ten elements, 4.5 M.-A.
(β) Muscles of the nerv. radial. r.: C. (contraction) at about 80 S.-D.

(b) Galvanic examination (normal electrode, Erb):

(α) Nerv. rad. r. CaSC, 2.3 M.-A.; l. CaSC, 6.0 M.-A.[3]
(β) Muscles (extensors and supinator):
r. at 3.5 M.-A.; CaSC > AnSC, C., short;
l. at 7.0 M.-A.; CaSC > AnSC, C., short.

3. What to Observe in Determining the Electrical Reaction.—We examine in two main directions: (a) the quantitative excitability or degree of excitability of the nerves and muscles; (b) the qualitative excitability of the muscles under galvanic stimulation.

(a) **Quantitative Excitability.**—Its diminution in the most marked degree—namely, loss of excitability—is easily recognized. To the record is always to be added: "Lost when the coils of the induction apparatus were separated to a distance x, or for a current of x M.-A." On the other hand, it is difficult to define the limits between the normal and pathological in simple diminished or increased excitability, particularly of the nerves. We can take different ways to arrive at a conclusion in this regard:

(a) We compare the two halves of the body. This is very much the more certain way, but of course is only applicable in cases of unilateral disease. Normally, the differences between the two halves of the body are very slight. The *maximal differences* for the nerves and with the galvanic current, according to Stintzing (58 healthy persons; Stintzing's normal electrode of 3 sq. cm.), are:

Ram. frontal. N. VII.	0.7 M.-A.	N. radialis	1.1 M.-A.
N. accessorius	0.15 "	N. peroneus.	0.5 "
N. medius	0.6 "	N. tibialis.	1.1 "
N. ulnaris 2″ above the olecranon	0.6 "		

For *faradic excitability* the difference for the two sides of the body, at least for the four pairs of nerves that come especially into consideration, rami frontal. (facial.), N. accessorius, ulnaris, peroneus (see below), is, according to Erb, scarcely ever greater than 10 mm. separa-

[1] See under Reaction of Degeneration.
[2] Fully expressed: right radial nerve, minimal contraction at 120 mm. spiral distance.
[3] This is the minimum, which must be given. A complete statement would be, if noted: for instance, r.: CaSC, 2.3 M.-A., AnSC, 3 M.-A., etc.

tion of the coils of his Dubois induction apparatus; according to Stintzing, the maximal difference of all the pairs of the body that are accessible for examination is 15 mm.

A difference which approaches this maximal difference must lead one to think of a pathological condition; a difference that is materially greater is certainly pathological. But whenever a difference is found we must always consider whether the two homonymous nerves are situated exactly alike (malformation of the bones, etc.).[1]

(β) We are to observe the relation which exists between the irritability of the N. frontalis (facialis), accessorius, ulnaris (at the elbow), peroneus, according to Erb's method.

These nerves, but especially the ulnaris and peroneus, show only slight differences in health, as the following table, taken from Erb's *Handbook*, shows:

Faradic Current.

1. Healthy person, mechanic, age thirty-eight years:

	Distance of coils in mm., minimal contractions.		Variation of galvanometer (old one), 10 elements.	
	r.	l.	r.	l.
N. frontalis	165	166	18°	19°
N. accessorius	172	177	16°	15°
N. ulnaris	159	158	6°	6°
N. peroneus	160	163	5°	9°

2. Healthy person, laborer, age twenty-four years:

	Distance of coils in mm., minimal contractions.		Variations of galvanometer (old one), 10 elements.	
	r.	l.	r.	l.
N. frontalis	195	192	17°	17°
N. accessorius	187	182	10°	9°
N. ulnaris	135	185	6°	10°
N. peroneus	180	180	5°	5°

Galvanic Current.

Healthy men, thirty-eight and twenty-four years of age. (Normal electrode, 10 sq. cm.):

	Occurrence of the first CaSC.		Occurrence of the first CaSTe.	
	r.	l.	r.	l.
N. frontalis	1.4 M.-A.	1.2 M.-A.	8.0 M.-A.	8.0 M.-A.
N. accessorius	0.5 "	0.5 "	4.0 "	4.0 "
N. ulnaris	0.4 "	0.4 "	6.0 "	5.5 "
N. peroneus	1.5 "	1.5 "	7.0 "	7.0 "

By studying these tables we ascertain from them the relation between these four pairs of nerves as to the extent of their irritability, and

[1] See above.

it is possible to recognize with greater certainty a bilateral variation, especially of the ulnar or peroneus nerves.

(γ) Lastly, Stintzing has given the "limits of value" for the irritability of nerves ascertained in the case of 58 healthy persons (Edelmann's galvanometer, normal electrode 3 sq. cm). But these figures are only of value for Stintzing's normal electrode. They are the following:

R. front. N. fac.	0.9–2.0 M.-A.	N. ulnaris	0.2–0.9 M.-A.
R. zygomat. N. fac.	0.8–2.0 "	2″ above the olecr.	
R. ment. N. fac.	0.5–1.4 "	N. radialis	0.9–2.7 "
N. accessorius	0.1–0.44 "	N. peroneus	0.2–2.0 "
N. medianus	0.3–1.5 "	N. tibialis	0.4–2.5 "

In individual cases, however, Stintzing has found still smaller or larger figures. As they are to be regarded as exceptions, he calls them "extreme values." Possibly some of them are of a pathological nature.

Except in the reaction of degeneration[1] the quantitative irritability of the muscles very often goes quite parallel with that of the nerves. We can endeavor to determine this by estimating it.

(*b*) **Qualitative Irritability of Muscles from Galvanic Stimulation.**—Although with respect to the nerves in general we are only interested in the strength of current required to produce the first occurrence of CaSC and CaSTe, since the law of contraction of the nerves is that normally the character is almost always lightning-like in the direct galvanic stimulation of the muscles, two important variations come into consideration: they concern *the character* of the contraction and the relation of the contractions to each other, and, further, *the law of contraction*, and particularly *the relation between CaSC and AnSC.* But the first point of view is much the more important.

There are two classes of pathological galvanic muscular reactions: 1, *the reaction of degeneration* (RD), the exclusive attribute of degenerative-atrophic paralysis; 2, *the myotonic reaction*, which occurs solely in Thomsen's disease.

1. Reaction of Degeneration (RD).—It is necessary to remember that normally the contractions which follow direct galvanic stimulation are either just as short and lightning-like as those resulting from irritation of the nerve, or at any rate only a trace slower, which slowness is relatively most frequently noticed with AnSC. Notice further that normally with a medium strong current, one which suffices to produce CaSC as well as AnSC, we find the CaSC greater than AnSC (CaSC > AnSC).

By *reaction of degeneration* (RD) is understood a complex phenomenon whose most striking peculiarity is a slow, vermiform contraction in direct galvanic stimulation and then by the occurrence of abnormally pronounced AnSC compared with CaSC. These anomalies are associated with diminished or even complete loss of irritability of the nerves for both currents, and corresponding behavior—that is, diminished or lost irritability of muscles—for the faradic current.

The slowness of the contractions produced in muscles by the con-

[1] See this.

stant current, in pronounced cases, is most extraordinary, catching the eye at once. The contractions of the muscles are actually vermiform, the movements in the respective limbs are correspondingly slow; the contractions may be quite extensive, but the natural force is always diminished. Reaction of degeneration (RD) is most easily recognized when combined with increased galvanic muscle-irritability, which is particularly the case in acute and anterior horn paralysis setting in acutely and affecting whole muscles or groups of muscles. The beginner is frequently astonished at the contrast between the lost or much diminished nerve irritability and the increased muscle irritability.

It is more difficult to recognize correctly reaction of degeneration (RD) if either the muscle irritability is on the whole much lowered, as it is especially seen in older paralysis, or if it occurs in muscles some parts of which react normally while other parts give RD—the normal and diseased bundles being mixed. The latter is a peculiarity of disseminated paralysis—as, for instance, spinal progressive muscular atrophy and some forms of neuritis.

Where reaction of degeneration (RD) is just developing, or at any rate where it is not pronounced, there AnSC is sometimes distinctly manifested by greater slowness than CaSC. Moreover, in such cases we often observe that weak muscular contractions better disclose the slowness than stronger ones.

If we remember that sometimes also in health the direct galvanic contraction lasts a somewhat shorter time, that is, executes its motion somewhat slower, than the contraction produced from the nerve, from this the important conclusion is drawn that slight traces of RD are sometimes not easily recognizable. In fact, for this considerable practice and experience are needed.

Increase of AnSC against CaSC is manifested by the circumstance that AnSC occurs either with currents of equal strength or even with weaker currents than CaSC; therefore, CaSC = AnSC, or even AnSC > CaSC. But since also normally AnSC produced directly is occasionally found to be equal to CaSC, and even greater than the latter, this peculiarity of reaction of degeneration (RD) is less positive for diagnosis.

Complete and *partial reaction of degeneration* are expressions used to designate the condition where, together with the existence of direct galvanic reaction of degeneration, the irritability of nerves and the faradic irritability of muscles is completely lost or is only diminished.

From what has been said we make the following schemata:

(*a*) *Complete RD.*—The electrical examination gives the following results:

Faradic:

nerves: I = 0; that is, irritability (I) lost;
muscles: I = 0; that is, irritability lost.

Galvanic:

nerves: I = 0; that is, lost;
muscles: slow, tonic, vermiform contractions:

the quantitative irritability about normal or increased or diminished; AnSC occurs with a less strong current than the CaSC, and with a

less strength of current from which both take place AnSC is greater than CaSC: AnSC > CaSC.

(*b*) *Partial EaR.*

Faradic:

nerves: diminution of I;

muscles: diminution of I.

Galvanic:

nerves: diminution of I;

muscles: RD as above.

For more ready comprehension we add here two curves from Kast, which graphically exhibit the normal muscular reaction and the RD.

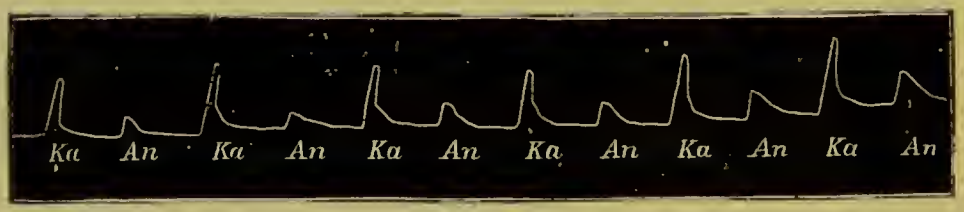


FIG. 177*a*.—Healthy young girl; stimulation of the muscles in the region of the peroneus; 33 cells. Ka = CaSC; An = AnSC (after Kast).

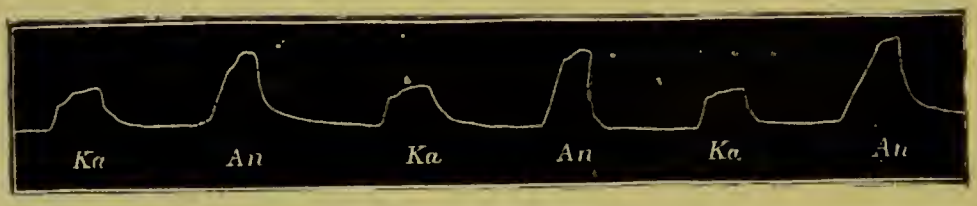


FIG. 177*b*.—Case of poliomyelitis anter. chronic, same muscles as above; 40 cells. Contractions tardy, AnSC > CaSC (after Kast).

Course of RD.—RD is the pathognomonic sign of those changes which take place in muscles or motor nerves and muscles when they cease to stand under the peculiar trophic influence of their anterior horn ganglia—those alterations we designate as degeneration of the nerves and muscles. This degeneration can be most beautifully studied by the electrical phenomena if a nerve-trunk is, at some place, suddenly interrupted throughout its whole transverse section. Whenever there is such an interruption there is manifest a complete separation of the portion of the nerve of the muscles located peripherally from the anterior horn, which must inevitably lead, not only to paralysis, but also degeneration of the portions cut off, and with it RD. But now the case can either proceed so far that there is a permanent interruption at the injured spot which results in complete atrophy of the nerves and muscular fibers, or, after a time, the conduction at this place may be restored; and in the latter case there is a return of the tissues of the nerves and muscles to the normal condition; that is, there is regeneration of them. Now, according as the degeneration of the nerve (muscle) is complete on the cross-section or only partial, further, according as the degeneration is complete, it results in atrophy (*i. e.* transformation into connective tissue), or it again regenerates and returns to its normal condition, the RD shows a definite result as

such, and also in its temporary behavior with reference to the ability to use the muscles within a given number of weeks or months. This course of RD in its relations to the alterations of the nerves and muscles is so well known that it can be used for a retroactive conclusion in regard to the latter.

It is to be noted that RD does not occur with *primary* disease of the muscles or with central diseases situated above the trophic centers.[1]

As regards peripheral paralysis the different forms of RD have their separate diagnostic significance. The prognosis for restitution is unfavorable in proportion to the extent and completeness of the RD.

A form of paralysis which occurs in all forms and degrees of severity is the so-called *rheumatic facial paralysis.* In this Erb, for the first time, recognized the relation of reaction of degeneration (RD) to the course of paralysis. We introduce here the schematic representation which he has given of its course.

Fig. 178a gives a representation of complete RD with reference to motility, and faradic and galvanic irritability of the nerves and muscles; and over it are given the designations of the simultaneous histological changes. The figures above the abscissæ signify the duration, and the height of the ordinates of the different curves indicates the degree of irritability. Where the respective curves sink below the abscissæ the irritability is extinct. The line of galvanic muscular irritability is wavy so long as the qualitative changes exist—that is, RD slowness of contraction and predominance of AnSC.

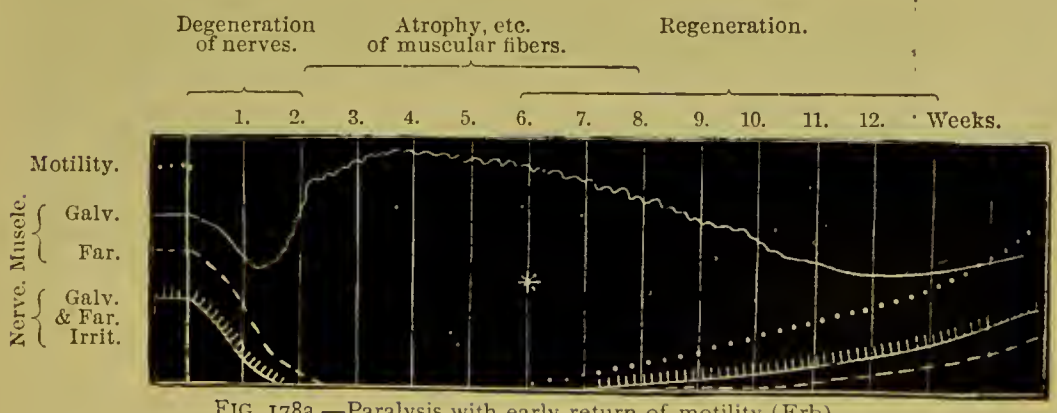


FIG. 178a.—Paralysis with early return of motility (Erb).

The motility is quickly completely lost; soon after the nerve irritability and the faradic muscle irritability diminish and are extinct after a fortnight. The galvanic muscle-irritability at first diminishes, but then with the signs of RD greatly increases. The first trace of motility appears at a time when there is still complete RD. One week later the faradic and galvanic irritability of the nerves reappears; hence there now is partial RD; three weeks later the slowness of the contractions begins to disappear. Diminished irritability of the nerves and motility continues a still longer time.

With reference to time the condition is like that in Fig. 178a. Here also for some time there is a partial RD. All the evidences of regeneration return later.

[1] See p. 484.

Motility, irritability of the nerves, and faradic muscular irritability do not return. Reaction of degeneration exists first, with increased muscular irritability. The galvanic muscular irritability in the course

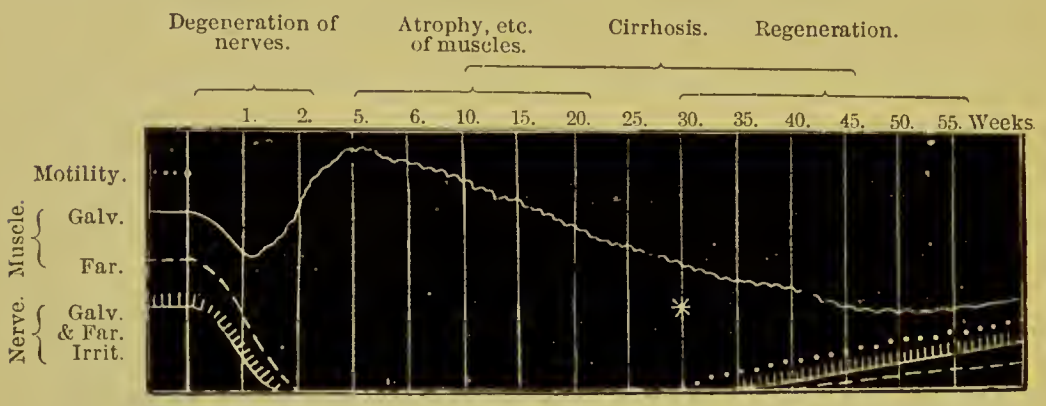


FIG. 178b.—Paralysis with later return of motility (Erb).

of some months becomes *nil;* the contractions, so long as they are still possible, are slow.

The faradic and galvanic irritability of the nerves and faradic

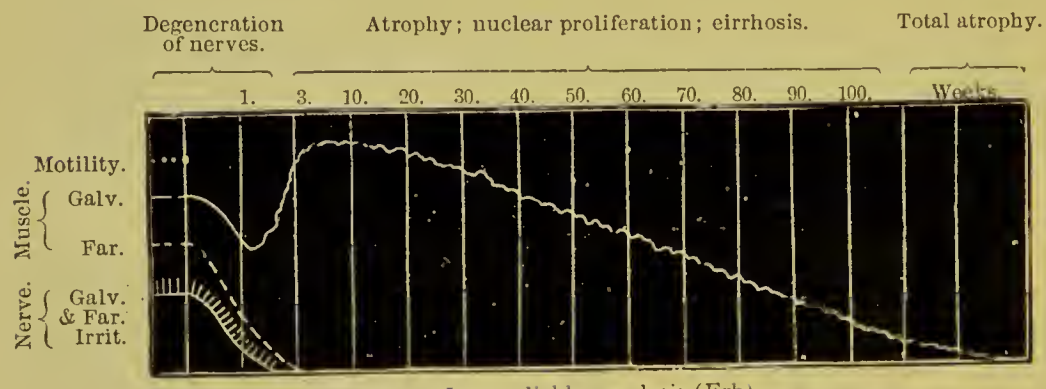


FIG. 178e.—Irremediable paralysis (Erb).

irritability of the muscles diminish only to a slight degree. Motility returns again quite early.

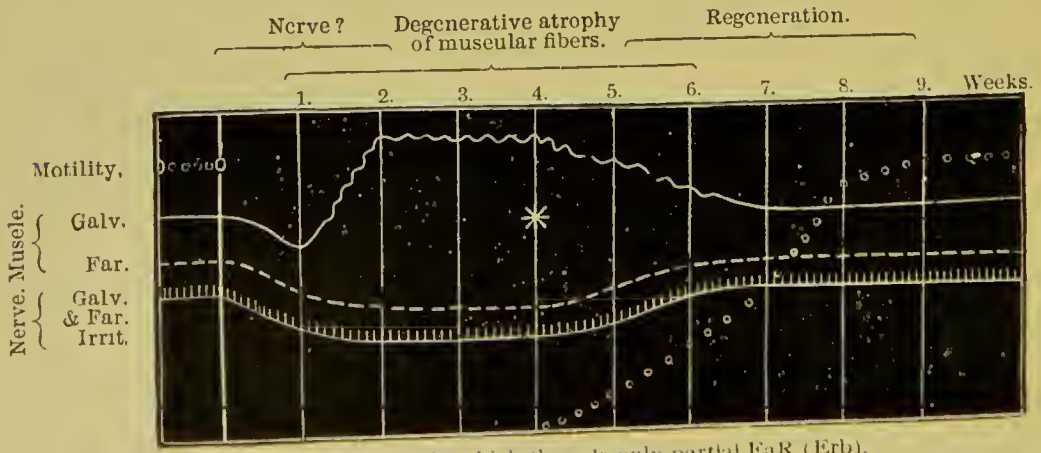


FIG. 178d.—Paralysis in which there is only partial EaR (Erb).

Varieties of RD.—(*a*) Partial RD is necessarily accompanied with slowness of contractions (which are also indirect—Erb). Not only the contractions which occur with direct galvanic irritation of the muscles, but all contractions, including those also which occur with galvanic and faradic stimulation of the nerves and faradic stimulation of the muscles, are slow in their character. ["The faradic excitability of the paralyzed muscle undergoes a diminution corresponding to that of the nerve, but the galvanic excitability of the muscles manifests the quantitative and qualitative changes which are characteristic of the severer forms of the reaction of degeneration."]

(*b*) The AnSC of the nerves is slow, the CaSC is not (Löwenfeld), or the muscle has a slow faradic reaction, while the nerve does not respond at all (Stintzing); or the muscle has a slow, the nerve a prompt, faradic reaction, etc.

Stintzing and others, with the greatest pains, have recently undertaken to bring order out of this confusion with remarkable, although with few, results. However, no considerable progress in differential diagnosis and prognosis of the cases has resulted from these labors.

(*c*) *Mixed Electrical Reaction.*—We thus designate those electrical reactions which occur when a muscle is partly degenerated and partly normal, and a corresponding portion of the nerves is also sound and another portion degenerated. Then we find a diminution, but never a loss, of faradic and galvanic excitability of the nerves and of faradic excitability of the muscles. But the direct galvanic muscular reaction causes the greatest difficulties: the contractions are not exactly short, not altogether slow, AnSC = CaSC, here and there also shorter: it is hard to discover its significance. All of this is not easy to understand, because normal contractions are mixed with RD; especially difficult is it if, as is almost always the case, the excitability is lowered. The object is sometimes attained by making repeated, indeed daily, tests (when it seems that RD often becomes more distinct) by thorough examination of every part of the muscular system with weak as well as with moderately strong currents, and frequently changing the location of the indifferent electrode (which must always be done in such a way as to avoid exciting the nerves).

A single clear manifestation of RD in one muscle or in a bundle of muscular fibers will usually serve as an indication of the whole disease as degenerative atrophic paralysis. It is true that RD has twice been found in myopathic muscular atrophy in single muscles (Schultze and Zimmerlin). We (with Erb) do not share the opinion of Wernicke that this mixture is the single cause of every case of partial RD.

2. Myotonic Reaction (Erb).—*Myotonia congenita* occurs in the very powerful (hypertrophic) muscles which always exist with this disease; they show increased irritability and continuance of the contraction with the faradic current; with the galvanic test likewise there is increased irritability, but only contractions as the current is closed, and then extremely slow and continuing contractions with peculiar formation of furrows and depressions. Stable-acting currents (the stimulating electrode placed not upon the muscle, but on the vasti, for instance, near the patella) produce rhythmical wave-like contractions from the cathode toward the anode.

In one case Jolly found that if without greater pauses we irritate repeatedly by the galvanic and faradic current, the duration of the after-continuing contraction always becomes shorter; the after continuation finally disappears altogether. This is a very interesting analogue to the behavior of myotonic muscles in active contraction.

3. **Diagnostic Value of the Electrical Condition.**—(*a*) **Significance of Reaction of Degeneration.**—The reaction of degeneration (RD) occurs —1. In all paralyses produced by disease of the ganglion-cells of the gray anterior columns of the spinal cord or of the motor nerves of the bulb. 2. In all paralyses produced by disease of the anterior roots and of the motor fibers of the peripheral cerebro-spinal nerves, where the trophic influence of the anterior-horn ganglia fails on account of the interruption of the conduction, peripherally, in the nerve and muscle.

The reaction of degeneration (RD), therefore, is closely connected with degenerative atrophy of the muscles. Thus it occurs—in poliomyelitis acuta, chronica, spinal progressive muscular atrophy, amyotrophic lateral sclerosis, lesions of a section of the gray anterior horns from hemorrhage, tumors, etc.; bulbar paralysis; in traumatic lesion of the peripheral nerves; in neuritis of all kinds; in "rheumatic" paralyses; in primary multiple neuritis; in toxic paralyses and those that occur after infectious diseases.

The presence of RD points directly in opposition to disease within the sphere of the central neuron, therefore against cerebral paralysis, and to paralyses which result from lesion of the pyramidal tract in the spinal cord; further, against myopathic paralysis; lastly, against functional or hysterical paralysis.

Of course, the RD is to be regarded as contraindicating the diseases last named only with the reservation that there is no complication with the conditions first named. Of this character we, with others, consider also the condition of RD found by Schultze and Zimmerlin with myopathic progressive muscular atrophy [see previous page].

In harmony with the above principles, *partial RD* has exactly the same significance as complete. It occurs—1. In slight affections (as slight forms of rheumatic facial paralysis, slight paralysis of the arm from pressure). 2. In atrophic paralysis, which only affects a portion of the bundles of the muscular fibers, it is disseminated (especially frequent in spinal progressive muscular atrophy, amyotrophic lateral sclerosis, multiple neuritis), and hence as a *mixed reaction*.[1]

When *RD is absent*, sometimes it does not strictly show that there is no affection of the anterior horns or of the peripheral nerves; that is to say, it does not do so if we have to do with a disseminated disease (see Mixed Reaction). RD may be wanting when there is an existing peripheral paralysis if it is very slight (very slight pressure-paralysis of the N. radialis, which heals in three to four weeks).

RD in muscles that are not paralyzed is seen by itself in lead-paralysis and traumatic paralyses.

(*b*) **Significance of Diminished Irritability.** — Lessened excitability, especially of nerves, without RD occurs chiefly in myopathic mus-

[1] See above, p. 483.

cular atrophy (dystrophia musc., Erb), in muscular atrophy from disease of the joints, and in lesions of the spinal pyramidal tracts, especially if recent and very severe. Moreover, it is observed with multiple neuritis, arsenic-paralysis, alcohol-paralysis, bulbar paralysis, amyotrophic lateral sclerosis, etc., and here it is probably to be counted as mixed reaction.

An intermitting general paralysis, which lasts say for twenty-four hours, with complete or almost complete loss of all electrical reaction, has been observed by Westphal. Its nature is very problematical.

(*c*) **Significance of Increased Irritability.**—Increased excitability as manifested by early occurrence of CaSC and CaSTe, occurrence of AnOTe, is an extremely important sign of tetanus. Slight increase is observed in cerebral, spinal, recent neuritic paralyses, in progressive muscular atrophy of spinal origin (here a more considerable increase, and this in muscles that are still performing their function).

The increase of galvanic excitability of the muscles with RD, as well as of the faradic and galvanic irritability of the muscles with myotonic reaction, does not belong here.

For Myotonic Reaction, see above, page 483.

5. Mechanical Excitability of Muscles and Nerves.

1. Upon striking a muscle with a percussion hammer we see that a short contraction occurs, like a CaSC with a tolerably weak current. We find these contractions increased and usually quite decidedly slow in those muscles which show electrical RD—"mechanical RD." If distinctly present, this shows the same thing as the electrical RD; but, often enough, it either fails or is not distinct, while the electrical examination proves the existence of RD.

Increased mechanical excitability with energetic but slowly declining and prolonged contractions (to as much as thirty seconds, Erb) are peculiar to myotonia congenita [see page 483].

For those who are experienced mechanical excitability is not without its value as a preliminary starting-point. But it cannot be a substitute for the electrical test.

2. *Idio-muscular contractions* are transverse prominences which appear locally at the spot where the muscle is struck—thus far, without any diagnostic significance.

3. *Mechanical excitability of the nerves* (striking upon the trunk of the nerve at the point of electrical stimulation) has individual differences. In many healthy persons mechanical irritation does not cause any contraction at all. The mechanical excitability of the nerves—but not of the muscles—is very much increased in tetanus. In this disease, on percussing the nerves of the extremities and the facial nerve, we almost always see not only very strong contractions in the respective muscles, but in most cases even a quick, somewhat vigorous movement of passing the hand downward at the posterior part of the cheek, diagonally to the branches of the pes anserinus, suffices to produce an effective, very short contraction of all the mimic muscles of that half of the face. This phenomenon, called the "facial phenomenon," in combination with the other phenomena of

tetanus, has great significance; by itself, however, it is not decisive, since it occasionally occurs also in other neuroses, especially in neurasthenia, and even in healthy persons of any age, particularly in children.

4. Charcot has discovered that a peculiar form of over-excitability of the nerves and muscles is characteristic of the *lethargic stage of hypnosis* in very hysterical persons—pressure upon the nerve or muscle causes contracture.

We mention here, further, the peculiar and obscure phenomenon of *paradoxical contractions* (Westphal). In passive dorsal flexion of the foot there occurs a tetanic contraction of the tibialis anticus which lasts from a few seconds to several minutes; the tendon of the muscle becomes prominent; the foot, even when it is no longer held, remains dorsally flexed. It frequently occurs in connection with increased tendon reflex.

6. Co-ordination and Ataxia.

In all motions there is necessarily a more or less complicated concurrent action of a number of muscles. For example, in order to seize anything with the hand, not only are a series of muscles of the arm, hand, and fingers moved, but at the same time, or a minimum of time before, the scapula, as a fixed point for the arm, must be steadied; moreover, from the free attitude of the body, the shifting of the center of gravity brought about by the motion of the arm must be equalized by the contraction of the muscles of the trunk and legs, equilibrium must be maintained—a proceeding which, it is evident from what has just been said, cannot be sharply defined. Hence, in order that the hand may attain its object, and in order that it may attain it in the shortest way and with a steady motion, a very exactly defined number of muscles must contract at the right instant and with the finest adjustment of energy. This correct selection of muscles and their regulation as to time and gradation of activity are designated by the term *co-ordination*. Originally this is acquired by practice by means of conscious and unconscious direction of our motions; and it is preserved by an oversight which is continually becoming less conscious and more unconscious, and which all our motions acquire.

Children at first, for all intentional movements, are ataxic in grasping things as well as in walking, etc. But also in later years co-ordination for new, hitherto untried movements must be acquired: the more complicated they are, the more practice they require. Learning to play on musical instruments, to handle fire-arms, etc. are examples of this. The acquired co-ordination in walking can be partly lost again by want of use of the legs and debilitation from long-continued severe sickness, as in typhoid fever.

The processes for acquiring and for maintaining co-ordination are certainly very diversified. Co-ordination will be acquired by the corrections which will be suggested by sensible irritations of all kinds, caused by the motions that are made and conducted to the central organs: the eye sees, the ear (of the violinist, for example) hears the motion itself or its effects, then the sensibility of the skin and the

whole totality of deep sensibility furnishes information, and the correction depends upon the sense of power of the muscles, which gives unconscious information regarding the intensity of the work accomplished each time by the muscle. In this acquisition of co-ordination the conscious will participates in many ways; on the other hand, in maintaining co-ordination it recedes very extraordinarily, and gives place to an unconscious influence of the motions by centripetal influences. But, if necessary, it may at any moment take hold, and even with an effect contrary to that intended, in that the unusual, acquired, hence now again, new agent of the regulation of the will disturbs the co-ordination, which went on successfully before unconsciously. A person says, "I will make it particularly beautiful," and just at that instant he becomes awkward. This happens not only with nervous and embarrassed people, but also with those who are very calm: under the control of the will they suddenly perform a motion which has long been automatically made.

Now, there is scarcely any doubt as to the nature of the centripetal influences, but where and how they bring their influence to bear upon the motor tract is very far from being clear. Voluntary motions certainly proceed to a certain extent from regulation derived from the cortex (where the complex motions, like those for speech, must exist), but certainly still other portions of the brain, which probably act as reflex centers, have an influence upon this regulation (thus especially the cerebellum for the motions of the trunk and legs); and, lastly, no doubt the gray anterior horns have a part in directing the continuity of motion: they preside over the tonus of the muscles, the antagonizing tension constantly in action during activity; they are the seat of tendon- and skin-reflexes. That all these things have an influence upon the continuity of motion cannot be doubted. But, likewise, there is no doubt that the various centripetal influences upon co-ordination to a very great extent may act vicariously for one another, so that when there is the loss of the conscious skin and muscular sensibility or in the disappearance of centripetal stimulation, they call forth the muscular tonus, the more attentive regulation of the cortical innervation (with the assistance, for example, of the eyes) replaces the loss of constancy; that, on the other hand—for instance, in the case of the blind—the exquisite superficial and deep sensibility, conscious as well as unconscious, must become prominent. But now, if co-ordination can no longer be maintained, then with its disturbance there occurs *ataxia*. It is clear from the foregoing that ataxia may exist at the same time with perfectly normal vigor; indeed, it has nothing whatever to do with native strength.

Ataxia shows itself according to its degree only with delicate, or it may even with gross, actions. It usually occurs as an excess of innervation in the sense of directing motion or as a want of restraint (tabes)—swing of the legs in walking, putting the feet down as if stamping, or only a clumsy way of moving the feet when turning around (in closing the door of the consulting-room); thus, on account of the uncertainty, the legs are spread out in standing and walking; impossibility of describing a circle with the foot when lying in bed, inability to exactly place the heel upon the knee of the other leg; when endeavoring

to take hold of anything, the hand misses it, as in the effort to take hold of one's own nose, in executing with the hand the finer movements of all kinds. In other kinds of ataxia there are other kinds of uncertainty, without this character of missing the mark, or the ataxia of the legs and trunk manifests itself by reeling. The control of the eyes sometimes diminishes the ataxia, sometimes not; the first is often the case in tabes. Most ataxic patients accordingly show a noticeable inward consciousness with every, no matter how, ordinary voluntary motion (as walking), quite in contrast with persons in health [see *Muscular Sense*, page 444*f*].

Ataxia occurs—(*a*) In cerebral affections, and particularly those of the cortex; here with paresis, confined to a limb or one-half of the body; with lesions of the vermiform process of the cerebellum, of the crura cerebelli, and of the pons and the corpora quadrigemina; and, lastly, in individual cases in ordinary hemiplegia if there is slight spasm; (*b*) especially in tabes, where ataxia is the most important symptom, sometimes after disease involving the whole thickness of the spinal cord; (*c*) rarely, and generally to a slight degree, in diffuse peripheral neuritides; (*d*) rarely as a highly-developed disturbance, though very frequently ataxia is distinctly recognizable in some persons after long confinement to the bed and especially after acute infectious diseases. Co-ordination is here temporarily and only partly lost.

7. Spasms of the Voluntary Muscles.

We combine under this designation all those pathological motions existing outside of the influence of the will; so we must go very much beyond the popular literal idea of "spasms." But this cannot very well be avoided unless we purposely wish to divide the subject very minutely. First, then, a few general remarks:

Tonic spasms are those lasting some time—from minutes to days and weeks—and are symmetrical. *Clonic spasms*, on the contrary, are contractions of short duration, followed by relaxation of the affected muscles. All, with the exception of some forms of trembling, exhibit phenomena of irritation derived from the nervous system, and, in fact, chiefly from the cortex, pyramidal tracts, the anterior horns of the spinal cord; some probably also from the peripheral nerves (also from the muscles themselves—paralysis agitans, contractions of fibrillæ). The pathological irritation is probably generally a direct one, but certainly also partly reflex; and, indeed, there is no doubt that the same kind of spasm may be caused by direct as well as reflex influences—as partial traumatic and reflex epilepsy. Many kinds of spasm consist of motions that are always similar—many combined from a few and sometimes from a great many.

Spasms are partly the intrinsic element of the given disease, the thing of which the disease consists; partly they are a symptom; and then, again, they may be a local sign or local symptom; that is, they may point directly to the seat or point of origin of the disease. Often we must determine other phenomena (as paralysis, etc.) for the purpose of discovering the point of origin.

With certain spasms, especially those that are paroxysmal and

general, the condition of self-consciousness at the time of the attack is of great diagnostic importance. Also we often have to consider the general mental condition, for many cases of convulsions lead us over into the territory of psychiatria.

We now only mention the different kinds of spasm:

Trembling (tremor) consists of unproductive motions, often only to be seen by close observation, rapidly following one another. We recognize them partly by observing the limb when at rest, partly when the hand is extended, or is holding a glass of water, and also by the handwriting.

Graphic representation shows that the different forms of tremor differ in the form, frequency, and rhythm of the contractions. Trembling is physiological with bodily exertion and with mental excitement, and it is sometimes constant, even with persons in good health. Upon the borders of the normal stand the tremors of the aged, *tremor senilis*. *Alcoholic tremor*, especially of the extremities and tongue, occurs with the passing away of the effects of the indulgence, or when it is declining; the *tremor saturninus*, the tremor which affects morphia-habitués when they abstain from it, that with *morbus Basedowii* (generally very fine, rapid movements, sometimes also coarser contractions), and the tremors of nervous individuals, are the finer kinds of tremors.

The tremor of *paralysis agitans* (especially of the extremities, but also of the head) manifests itself by a symmetrical rhythm, by a very characteristic position of the hand and fingers ("pill-maker"). It ceases when voluntary motions are made, especially if vigorous, but sometimes even when writing.

On the other hand, the *intention tremor* occurs only with voluntary motions, in that toward the end of the motion it becomes stronger; it stops as soon as the patient is quiet. It is an important symptom of multiple sclerosis; it occurs, however, as *tremor mercurialis*. In many cases it is difficult to distinguish it from ataxia.[1]

Between "tremor" and "clonic spasms" it is not possible to draw a precise distinction. The designation *shaking-spasm* is used for the transition forms of both. The prominent transition forms of this kind of tremor are those shiverings which begin with fine tremors, becoming constantly coarser with cooling off, and with rapidly-rising fever; with hysteria there are conditions that resemble tremor. Likewise is to be mentioned the quaking which occurs with marked active spasm of the legs, as especially takes place sometimes after mechanical irritation; foot-clonus, particularly, often shows these transition forms very beautifully.

In the foregoing we have not distinguished between the tremors of spasm and those of paralysis, because in regard to most kinds of tremors it is not yet clear to which of the two classes they belong.[2]

Fibrillary Contractions.—These are contractions in individual coarse or fine bundles of muscular fibers which ordinarily do not produce motion in the limb; only in individual cases, however, we can observe a very diminutive motor effect. They are easily recognized by observing the muscle. In health they are often excited (with great individual differences) by the cooling of the skin; but they also occur with

[1] See above.

[2] Regarding this, see the several special works.

atrophic paralysis, and very abundantly, and hence are not without diagnostic value, in spinal progressive muscular atrophy.

Clonic spasms rarely occur by themselves, but they more frequently accompany epileptic and other attacks of convulsions.[1] We sometimes observe them isolated in local affections of the cortex of the brain;[2] but also in other localized cerebral diseases, and in myelitis transversa, as single brusque bending motions of the legs, generally both legs together—probably of reflex origin.

Tonic spasms, by themselves, occur most frequently in the form of active spasms,[3] in lesions of the pyramidal tracts, and with hysteria. Moreover, they occur in tetanus, and in these forms: as masseter spasms in trismus (this latter also by itself); as rigidity of the face, *risus sardonicus;* extension of the vertebræ: *rigidity of the neck* and *opisthotonos*, and in spasms of the legs in the state of extension. Moreover, tonic spasm of the muscles occurs when first moving them after long rest, and as a prolonged condition after voluntary contractions in *myotonia congenita;* also, occasionally, as bending and adduction spasms of the arm and hands in *tetanus;* as the tonic form of *writers' cramp*, although seldom purely as such, generally with slight contractions mixed with tremor; and in the first stage of *epileptic attacks* (see below).

Epilepsy.—In genuine epilepsy, generally though not always the convulsions pursue a typical course: after certain subjective warnings (*aura*), or without these, there is a sudden loss of consciousness, ushered in with a cry, and immediately the patient falls. Then there is a short *tonic spasm* of all of the voluntary muscles (more especially of the extensors of the arms, legs, vertebræ, but the hands are closed and the thumb is grasped by the fingers); then there is *clonic spasm*, with great vigor of all the muscles of the body, including the muscles of the eyes, tongue, etc.; after a few minutes there follows, either gradually or suddenly, a period of relaxation with continued loss of consciousness—*post-epileptic coma.* During the attack the tongue is often bitten, involuntary discharges take place, and, from the interference with respiration, marked cyanosis often occurs.

It is very important to make a differential diagnosis between genuine epilepsy and *symptomatic convulsions*, which often very much resemble the former. The latter occur in all manner of anatomical diseases of the brain (regarding partial epilepsy in disease of the cortex of the brain, see below), as traumatic and reflex epilepsy, as epileptiform spasms in uremia, these latter also as *eclampsia gravidarum.*

There occur in children, upon slight provocation, epileptiform or eclamptic attacks during dentition, from intestinal irritation from worms, in the beginning of acute infectious diseases, as scarlet fever, measles, pneumonia, and in the beginning stage of acute poliomyelitis and encephalitis.

It is generally very difficult to form an opinion regarding spasms from the anamnesis. Here we must be very cautious in arriving at a diagnosis.

Partial or Jackson's Epilepsy, Dissociated Spasms.—In this there are epileptiform convulsions which are limited to an extremity or to the

[1] See below. [2] See below, Partial Epilepsy. [3] See above, p. 456.

facial muscles of one side. They are an almost infallible sign of disease located in a corresponding part of the cortex of the brain, and also are accompanied or followed by paresis, increased tendon-reflex, and sometimes by disturbance of the sensibility of the affected limb (*monoplegia*). The convulsions may be unilateral or even general, but they always manifest themselves as originally partial-epileptic by beginning in the affected limb, recently designated by many as the one "primarily having spasms."

Hysterical convulsions (attacks of hystero-epilepsy) sometimes have a great likeness to epilepsy, yet almost always the motions may be distinguished in that they are more wide-reaching [and tumultuous], and more than all by the fact that they partly manifest co-ordinated motions or remind one of them. Motions such as we see made by a person senselessly furious or an unruly child are not at all infrequent; especial manifestations are fits of laughing, shouting, weeping, coughing.

The most important mark of difference between hysterical and epileptic spasms in doubtful cases is that in the former there is almost never an entire loss of consciousness; very often it remains quite intact; and the absence of involuntary discharges (urine, stool, in males also of semen), as is not infrequent with genuine epilepsy; lastly, the tongue is not bitten and there is reaction of the pupil during the attack.

Gross [Severe] Hysteria.—The attack of hystero-epilepsy may pass into a second stage ["phase des grand mouvements" of the French] of contortions and excessive movements—among others, especially that of the "arc de cercle" (head bent backward, boring into the pillow; the trunk bent as in opisthotonos), which may last for hours, is a characteristic manifestation; then there may follow a third stage, which is either quiet or may be excited (delirium)—the stage of hallucinations and of emotional attitudes. The stages may occur singly.

Besides what has already been described, it is important for diagnosis that the patient should manifest *hysterical signs* (stigmates hystériques) in the form of sensory anesthesia, especially a concentric limitation of the field of vision; also, hemianesthesia; lastly, hysterogenous zones; that is, hyperesthetic regions of the body (ovaries, testicles, circumscribed portions of the skin), the irritation of which by pressure sometimes causes an attack or is associated with one.

Constrained Positions and Motions.—To the former belong drawing of the head or trunk to one side, so that the patient assumes the side position in bed (sometimes with the eyes fixed; déviation conjugee occurs with the other manifestations); to the latter belong the involuntary forward, backward, and movement in a circle (manègegang). Both phenomena indicate a lesion of the vermiform process of the cerebellum or of the median crus cerebri.

With the constrained motions or "co-ordinated spasms" are also to be reckoned the gross motions previously mentioned under hysteria, as laughing, screaming, etc.

Chorea Minor.—This is the designation given to the very rapid, lightning-like, entirely irregular muscular contractions, which, on the one hand, produce restlessness of the limbs and of the face, and, on the other, divert the regular voluntary motions. They affect the head (face, tongue, masticating muscles), the muscles of the trunk, especially of

the shoulders and legs, and sometimes the glottis. They occur in all degrees of severity, from single weak jerks to the most extravagantly confused strong movements (folie musculaire). If the subject is embarrassed, especially if observed, frequently the contractions are increased. During sleep, but there may be difficulty in getting to sleep, the convulsions entirely disappear, excepting in particularly severe cases.

Chorea minor is not often purely one-sided: *hemichorea.* Hemichorea may occur either as the forerunner or as the result of hemiplegia when it indicates a lesion of the posterior section of the inner capsule or of the optic thalamus. Especially frequent are choreic or athetose motions[1] in the paralyzed limbs, with declining acute encephalitis in children (polio-encephalitis—Strümpell). Quite recently Flechsig has found both internal segments of the lenticular nucleus diseased in several cases of severe general chorea with delirium.

Athetosis [described by W. A. Hammond].—This designates peculiar, slow, and at the same time tolerably energetic motions, particularly of the hands, arms, shoulders, but also anywhere else. If the motions are somewhat quicker than, but resembling, those of chorea, they then form a transition to the latter. Athetosis, as well as chorea, is a disease in itself; hemiathetosis is observed in the same cerebral locations as hemichorea (which see). In the cerebral paralyses of children it is more frequent than hemichorea.

Associated movements are abnormal involuntary motions which take place, with the performance of voluntary motions, by contraction of muscles in regions which have nothing to do with the motions desired. We find them especially in cerebral, but also in spinal and even in peripheral, paralyses; hence they cannot be made use of as an aid in diagnosis. Sometimes we see them in the muscles of the limb which is being put in motion. Particularly frequent is a dorsal flexion of the foot when the leg is drawn up to the abdomen, as in hemiplegia, spastic spinal paralysis (Strümpell), or in the unilateral affections as synonymous associated movements of the sound side with those of the diseased side or of the diseased side with the sound side.

Catalepsy, cataleptic rigidity, flexibilitas cerea, is a peculiar increase of the tonus of the voluntary muscles of such a character that the limbs not only offer a very slight or feeble resistance in passive motion, but also remain in a given position, even when it is opposed to gravity, and this sometimes for an hour or more at a time. Catalepsy very rarely occurs in anatomical diseases, as tumors of the brain and meningitis; more frequently in hysteria, especially in hypnosis, and in certain psychoses, as *melancholia attonita.*

8. Voluntary Muscles, their Innervation, their Function, and the Diseases that Disturb Them.

1. **Muscles of the eye** (see Examination of the Eye).

2. **Muscles of the face,** supplied by the N. facialis:

M. frontalis draws up the brow and causes wrinkles across the forehead.

[1] See below.

M. corrugator supercilii draws the skin of the forehead over the root of the nose into folds.

M. orbicularis palpebrarum closes the eyes.

M. depressor nasi seu dilator narium dilates the nostrils.

M. levator labii superioris (proprius) and M. levator anguli oris lift up the upper lip and the corner of the mouth.

M. zygomaticus major raises and draws out the angle of the mouth.

M. buccinator makes the cheeks tense, holds open the pouch of the cheek when eating, prevents the distention of the cheeks when blowing or whistling (to a slight extent supplied by the trigeminus ?).

M. orbicularis closes the mouth; it is the chief factor in whistling, pronouncing the consonants *b*, *f*, *m*, *p*, *v*, *w*, the vowels *o*, *u* (greatly assisted by the levator menti).

Paralysis of the Facial.—The forehead is smooth and remains so upon the affected side when the effort is made to wrinkle it; the eye remains open and cannot be closed (lagophthalmus); the naso-labial furrow is obliterated; the angle of the mouth hangs down; the mouth, and often also the tip of the nose, are drawn toward the sound side; the effort to expose the teeth, as in cleansing them, makes very plain the defective elevation of the upper lip and distortion of the mouth. When blowing, the affected cheek is distended; on attempting to whistle, the lips are drawn to the sound side; if the paralysis is unilateral, the labials are generally, except in recent paralyses, pronounced distinctly; if bilateral, they cannot be. See, further, Soft Palate, Hearing, Taste.

Cerebral facial paralysis, from disease of the cortex, or in the course of the pyramidal tract, is usually distinguished from peripheral paralysis in a remarkable way: in the first place, the former almost never affects the whole facial of one side, but leaves untouched, or nearly so, the forehead and ocular facial. This may possibly result from the fact that on both sides the muscles of the forehead and for closing the eye are innervated from both hemispheres, because they are usually active on both sides simultaneously; furthermore, in cerebral facial paralysis the emotional innervation is generally preserved: the patient in vain tries to move the angle of the mouth on the paralyzed side when ordered to do so, and yet when laughing shows little or no difference between the two sides of the countenance. This is never the case in peripheral facial paralysis, for here, on the contrary, voluntary and emotional paralysis of course always go together. From this peculiarity of cerebral paralysis, however, it follows that the emotional facial tract in the brain is distinct from the voluntary. This assumption is confirmed by the additional fact that cases occur in which there is purely emotional facial paralysis, without visible paresis in repose, and without volitional paralysis. The anatomical conditions in these cases indicate that the optic thalamus and fibers of the corona radiata which originate from it come into consideration for the emotional facial tract further downward in the tract of the cerebral peduncle and tegmentum.

3. Muscles of Mastication, Tongue, Soft Palate, Pharynx.—Mm. temporalis and masseter (N. trigeminus branch III.) draw up the lower jaw and press the teeth together. Mm. pterygoidei effect the sideways movement (rotation) of the lower jaw.

Paralysis of these muscles will be recognized by the absence, upon one or both sides, of these motions; bilateral paralysis of the temporalis and masseter, by the dropping of the lower jaw. Palpation below the zygoma detects possible paralysis and atrophy of the masseter; above the zygoma, paralysis and atrophy of the temporalis is shown by its laxity.

We pass over the complicated arrangement of muscles which draw down the lower jaw, because paralysis of these muscles has not yet been sufficiently studied.

The tongue is protruded—that is, it is drawn forward by the two Mm. geniohyoglossi, which act somewhat convergently—and it is drawn back chiefly by the two Mm. styloglossi; M. hypoglossus principally draws it down. These and the inner lingual muscles produce the changes in the form of the tongue.

Unilateral Hypoglossal Paralysis.—When the tongue is protruded it deviates toward the paralyzed side, because the genioglossus of the sound side pushes it that way. Bilateral paralysis (generally atrophic) causes diminution of all the motions, even to their complete obliteration, difficulty in mastication and swallowing and in the formation of the consonants *c*, *d*, *g*, *k*, *l*, *n*, *r*, *s*, *sch*, *x*, *z*, and of the vowels *i* [*ē*] *e* [*ā*]. Unilateral paralysis produces all these disturbances to a slight degree, and they become less with habit. *Atrophy*, seldom unilateral, will be recognized by diminution in the volume, by wrinkles, and sensible thinness.

The soft palate derives its principal innervation from the sphenopalatine ganglion (N. petrosus superficialis major), and from the ganglion geniculi of the facial nerve. The fifth and the tenth and eleventh ganglia also take part.

Examination.—By inspection and phonation—*i. e.* by observing the voice and inspection, and by the swallowing of fluids.

Unilateral paralysis of the soft palate in paralysis of the facial located high up is manifested by deviation of the uvula toward the healthy side and depression of the arch of the paralyzed soft palate, both more distinctly in phonation. In the passive state the relaxed uvula may hang to one side, even when there is no paralysis. Sometimes the speech is nasal, and fluids may escape from the nose in attempting to swallow. Both symptoms are due to ineffectual closure between the nose and the mouth—pharyngeal space. In *bilateral paralysis*, especially with bulbar paralysis and as diphtheritic paralysis, the soft palate hangs down without any power to contract, and nasal utterance and the difficulty in swallowing are increased.

The pharyngeal muscles (Nn. X., XI.), with the aid of the tongue, accomplish the act of swallowing. When they are palsied this act is disturbed, and, from the lack of vigor and promptness in passing the food along, food easily enters the larynx; thus there is coughing in connection with swallowing. But if the patient is unconscious or at the same time there is disturbance of the sensibility of the larynx (N. laryngeus superior vagi), there may be no cough.

4. Laryngeal Muscles.—The muscles supplied by the laryngeus superior vagi are—depressors of the epiglottis; Mm. thyreo-epiglottici, aryepiglottici (if paralyzed: difficulty in swallowing), and the M. crico-

thyreoideus, tensors of the vocal cords by movement of the thyroid cartilage toward the cricoid cartilage (if paralyzed: hoarse voice).

N. laryngeus inferior (recurrent branch of the N. X., XI.): Mm. crico-arytænoid. postici dilate the glottis (if there is bilateral paralysis: inspiratory dyspnea, sometimes of the severest kind, with the voice unchanged or very slightly impure). Mm. thyreo-arytænoidei are the most important tensors of the vocal cords (if paralyzed: loss of voice and hoarseness). Musculi arytænoidei transversi et laterales: they narrow the posterior portion of the glottis. In isolated paralysis of the muscles the voice is very hoarse (as in catarrh, hysteria). Mm. crico-arytænoidei laterales: in connection with the preceding they narrow the glottis.

Complete paralysis of the recurrent: (*a*) unilateral (compression by aortic aneurysm, carcinoma of the esophagus, mediastinal tumors; bulbar paralysis): voice is hoarse, easily changing to the falsetto or little or even not at all altered; (*b*) bilateral (rare): there is complete aphonia, inability to cough.

The laryngoscopic examination is indispensably necessary, regarding which see Appendix.

5. Muscles of the Throat and Neck.—M. sterno-cleido-mastoideus (N. XI.) draws the head and face toward the opposite side and in the position of looking upward; both together somewhat bend the neck and push the head forward; or, if the head is the fixed point, they lift up the sternum or the clavicles, as in emphysema. The test of their function and recognition of their paralysis and spasm are easy. When both are paralyzed, the neck, and with it the head, incline backward.

The muscles that stretch, bend, twist the neck or the head (nervi-cervicales I.–IV.) maintain the head in the upright position. If they are weak or paralyzed it is impossible to hold the head up: it falls forward if it is not exactly balanced. This happens if the head is too heavy (*hydrocephalus*). Defective mobility of the head is more frequently caused by spasm or inflammation (stiff-neck, caries of the cervical vertebræ) than by paralysis.

6. Muscles of the Trunk.—Muscles that move the vertebræ (innervated by Nn. dorsales and lumbales):

Lumbar extensors and extensors of the lower vertebræ: M. erector trunci (sacro-lumbalis et longissimus) with bilateral action.

Bending forward: the abdominal muscles.

Bending of the lower vertebræ sideways: quadrati lumborum.

Twisting the trunk: semispinalis and multifidus.

Paralysis of the erector trunci: (*a*) Bilateral: the body is bent backward (lordosis of the lumbar, kyphosis of the upper thoracic, vertebræ, in such a way that the latter overhangs the sacrum; a plumb-line held from it falls behind the sacrum); the pelvis is tilted up, the knees are bent. (*b*) Unilateral: in standing a scoliosis of the lower vertebræ is convex toward the diseased side; on the other hand, there is a compensatory scoliosis of the thoracic vertebræ.

Paralysis of the abdominal muscles: marked lordosis of the lumbar and lower thoracic vertebræ, compensatory kyphosis of the upper thoracic vertebræ, but these are exactly vertical over the sacrum. There is marked inclination of the pelvis.

In paralysis of the extensors it is impossible to place the bent trunk in an unsupported upright position; it is accomplished by placing the hands upon the knees and thighs. If, in addition, there is paralysis of the glutei, especially of the gluteus maximus, then the patient can only rise from the floor by first getting down on "all fours," then pushing himself up with the hands from the floor, in order immediately to put them upon the knees and thus further support the body: this is his only way of standing up. In paralysis of the flexors it is impossible to change from the dorsal to the sitting position without assistance.

Opisthotonos is produced by spasm of the extensors, *emprosthotonos* by spasm of the flexors; unilateral spasm of the extensors causes scoliosis, convex toward the diseased side.

7. Muscles of the Thorax, Diaphragm, and Abdomen.—Here belongs most of what has already been said upon page 72, *ff.* There we learn regarding the ordinary and the auxiliary muscles of inspiration and the auxiliary muscles of expiration.

Paralysis of the diaphragm (phrenic nerve, chiefly from the fourth nerve of the [deep] cervical plexus) in perfect quiet may be entirely compensated by the thoracic muscles of inspiration, but otherwise every increased requirement for breath produces marked dyspnea; and this is exactly the case with respect to the vicarious action of the diaphragm when there is defective thoracic breathing. It will be understood, then, that *paralysis of the auxiliary muscles of respiration* has only a bad outlook for the breathing when it comes to such a pass that they must be called upon (see page 73).

Tonic and clonic spasms of the thoracic muscles of inspiration in tetanus and epilepsy at once cause severe cyanosis; in the first disease it may be fatal; also tonic spasm of the diaphragm interferes very much with breathing and may be dangerous to life. Clonic spasm of the diaphragm (*singultus, hiccough*) in a mild form is not infrequently seen; if it continues for hours and days, as it sometimes does in abdominal and cerebral affections, then from the disturbance of rest and severe pain along the line of insertion of the diaphragm it may bring about a serious condition.

By the contraction of the abdominal muscles the anterior abdominal wall is flattened, and thus the abdominal cavity is lessened; by the simultaneous contraction of the diaphragm there arises "the abdominal pressure," which is important in defecation, urination, and expulsion of the child in labor. The rôle of the rectus and obliquus externus as flexors of the vertebral column (when those of one side act alone the trunk is bent laterally forward over to one side) has been already mentioned, as well as their function in active expiration.

8. Muscles of the Upper Extremity.—(*a*) Muscles which move the shoulder-blade or fix it:

M. trapezius (N. accessorius for the most part) raises the shoulder-blade and draws it toward the middle line, both of these movements by its middle and posterior parts. The former chiefly lifts the acromion, the latter the inner upper angle. With its anterior clavicular portion it inclines the head obliquely backward and at the same time lifts the acromion. *Paralysis* of the trapezius permits the scapula to drop, to be drawn away from the middle line, and at the same time to

turn round so that its apex moves toward the spinal column (because the levator scapulæ holds up the upper inner angle). The shoulder sinks downward and forward; there is difficulty in raising the upper arm, because the scapula is not so perfectly fixed, and shrugging of the shoulders is restricted. From what has been said the test of its function is easy.

M. levator anguli scapulæ (N. dorsalis scapulæ from the cervical plexus and branches of this plexus) lifts the scapula by its inner upper border, with the tendency to turn the right scapula in the direction of the hands of the clock and the left in the opposite direction. Its *paralysis* can only be recognized, when the trapezius is paralyzed at the same time, by the complete inability to lift the shoulder.

Mm. rhomboideus major et minor (N. dorsalis scapulæ) draw the shoulder-blades toward the spinal column, and thus lift them in the same way as the levator scapulæ, and turn them in such a way that the lower angle of the scapula is nearest the spinal column. They fix the scapulæ, especially in backward motions of the arms and legs and when lifting weights. *Paralysis* [of these muscles] causes the scapula, and particularly its lower angle, to move away from the spinal column. Moreover, it is difficult to detect paralysis of these muscles when the trapezii are normal.

M. serratus anticus (N. thoracicus longus seu posterior (Henle), from the brachial plexus) turns the scapula in such a way that the lower angle moves outward, draws it somewhat away from the spinal column, and presses it against the thorax: it is an important fixation-muscle of the scapula when the arms are lifted. When the scapula is fixed (by the rhomboidei) it is a muscle of inspiration. *Paralysis of the serratus*, in the condition of rest, causes a slight elevation and rotation of the scapula, so that the lower angle stands out a little from the thorax and is slightly drawn toward the spinal column. The arm can be lifted to the horizontal sideways: this moves the inner border of the scapula close up to the vertebral column. It can only be raised higher by fixing the scapula in the same way as would be accomplished by the serratus. When the arm is moved forward the inner border of the scapula stands out like a wing.

(*b*) Muscles of the trunk and of the scapula [attached] to the upper arm:

M. deltoides (N. axillaris at the infraclavicular portion of the brachial plexus): the middle portion extends the arm outward from the body, the anterior portion raises it obliquely forward, the posterior portion obliquely backward. It raises it as far as the horizontal, beyond which, the arm being fixed by the deltoid against the scapula, it is raised by the rotation of the scapula. *Paralysis* is easily recognized: if the muscle is relaxed, there is subluxation of the humerus, particularly if at the same time the supraspinatus is paralyzed; if the deltoid is atrophied, the contour of the bones at the shoulder shows plainly.

M. supraspinatus (N. suprascapularis from the supraclavicular portion of the brachial plexus) assists the deltoid in raising the arm outward toward the front, and rolls it inward; it is also said to hold the head of the humerus in its socket when the arm is raised.

Mm. infraspinatus (N. suprascapularis) and the teres minor (N. axillaris) roll the upper arm outward.

M. subscapularis (N. subscapularis from the brachial plexus) is a rotator inward. *Paralysis* of a rotator allows the arm to rotate in the opposite course; in testing, we first make passive rotation, and, letting the arm fall, allow it actively to do the same thing while we oppose the rotation.

M. pectoralis major (Nn. thoracici anteriores of the brachial plexus) adducts the upper arm; when the arm is raised it moves it forward in the horizontal plane, draws the arm down when it is raised. Test: Have the upraised arm moved forward in a horizontal plane while we offer resistance.

M. latissimus dorsi (N. thoracico-dorsalis from the brachial plexus) draws down the arm when it is raised in exertion [it depresses it]; and draws it backward. When the arm hangs down it draws it backward and inward [toward the buttock]. Test: The arm is raised to the horizontal, and the effort is made to lower it while the movement is opposed. The teres major materially assists the latissimus; it is at the same time a rotator inward.

Mm. coraco-brachialis (N. musculo-cutaneus of the median) and anconeus longus (caput longum tricipitis; N. radialis), when the arm is drawn down by the latissimus and pectoralis, hold the head of the humerus up and firmly in its socket.

(*c*) Muscles from the upper arm to the forearm:

M. triceps (N. radialis) is an extensor of the forearm.

M. brachialis internus (N. musculo-cutaneus) is a simple flexor.

M. biceps (N. musculo-cutaneus) flexes and supinates.

M. supinator longus (N. radialis) flexes and pronates. This is proved by having the moderately pronated forearm flexed while the movement is resisted. If it is healthy, it rises like a hard roll on the outer side of the elbow-joint.

We here next mention the pronators: the pronator teres (it is at the same time a flexor) and quadratus, both supplied by the median nerve.

(*d*) Muscles which extend from the condyles of the humerus and the bones of the forearm to the hand and fingers, and the small muscles of the hand:

The extensor carpi radialis longus and brevis (N. rad.) + extensor carpi ulnar. (N. rad.) are elevators of the hand. The flexor carpi radialis (N. median) + flexor carpi ulnaris (N. ulnar.) are volar flexors of the hand; the palmaris longus (N. median) assists in this action.

The extensor carpi radialis longus + flexor carpi radialis adduct the hand in the direction of the radius. Extensor carpi ulnaris + flexor carpi ulnaris adduct the hand on the ulnar side. If the extensor carpi radialis longus acts alone, it raises the hand obliquely on the radial side, as the extensor carpi ulnaris does on the ulnar side.

Paralysis of the extensors of the hand (especially lead-paralysis, also sleep-paralysis of the N. radialis) allows the hand, when the forearm is pronated, to hang loosely. Paralysis of the abductors and adductors, and also paralysis of the extensores carpi radialis longus and carpi ulnaris alone, produce oblique position of the hand [paralysis

from the former giving a position opposite to that of the latter]. We test the individual movements by successively opposing them.

M. extensor digitorum (communis, indicator, extensor digiti V., all from the N. radial) extend the first phalanges.

M. flexor digitor. comm. sublim. (N. median) flexes the middle phalanges; M. flexor digitor. comm. prof. (N. median, the two ulnar bellies from N. ulnaris) flexes the terminal phalanges. Mm. interossei dorsales + volares (N. ulnaris) and Mm. lumbricales (N. median and ulnaris) flex the first phalanx, and at the same time extend the middle and terminal phalanges.

Mm. interossei dorsales alone abduct (spread apart) the fingers, volares alone adduct the (middle, third) finger.

Movements of the thumb: extensor pollicis longus (N. rad.) is essentially an extensor of both phalanges; extensor pollicis brevis (N. rad.) is an extensor only of the first phalanx. Adductor pollicis longus (N. rad.) abducts the metacarpus. Flexor pollicis longus (N. med.) flexes the terminal phalanx. At the thenar are the opposing muscles—abductor pollicis brevis, outer head of the flexor brevis, and the opponens pollicis (all from the N. med.). Adductors: adductor pollicis and the inner deep head of the flexor brevis (both N. ulnar.). These two and the abductor brevis flex the first and extend the terminal phalanx.

The adductor, flexor, and opponens act at the hypothenar, their names indicating their action. All are innervated by the N. ulnaris.

Characteristic positions of the hand and fingers: 1. In paralysis of the ulnar there is the clawing, clutching hand—*main en griffe:* the first phalanges are extended, the middle and terminal ones flexed (paralysis of the interossei), the thumb hangs helpless over the hand (paralysis of the adductor), the fingers are easily spread apart (action of the extensores digitorum). Thus the interosseal spaces on the dorsum are deepened, likewise the groove between I. and II. metacarpal bones (atrophy of the adductor pollicis, deep head of the flexor brevis and interosseous dorsi I.). The hypothenar is atrophic. 2. In paralysis of the thenar (deep median paralysis) there is the ape-hand: the thumb does not stand out opposing, but is parallel with, the other fingers.

Paralysis of the extensors of the hand causes apparent weakness of the long flexors of the fingers because the origin and insertion of the flexors are brought near together by the flexion of the hand at the wrist. Hence we must passively extend the wrist and then test the flexion of the fingers. For the same reason it is necessary, when there is *paralysis of the long extensors of the fingers*, to passively extend the first phalanx before testing the flexion of the middle and terminal phalanges.

Examination.—We observe the position of the hand for possible atrophy. Then we test extension, flexion, abduction, and adduction at the wrist—sometimes all of these—by resisting these motions; then the extension of the fingers; next the long flexors by "hooking" of the fingers; then let the patient make the separate motions of the interossei muscles; flex the first phalanx with the middle and end phalanges extended; then spread apart and close the fingers; test the muscles of the thenar and hypothenar by bringing the thumb and little

finger into contact; lastly, the examiner places his own index finger in the saddle between the thumb and the second metacarpus, while the patient makes simple adduction of the thumb, thus testing the power that is manifested. Pressure of the hand is a very practical way of making a general test of the long flexors and the small muscles of the hand. For such paralyses as are not wholly diffuse, but rather confined to individual muscles or groups of muscles (peripheral and certain spinal paralyses), it has value only as a preliminary examination. For various reasons we consider the dynamometer as an unnecessary apparatus and one that does not accomplish its purpose. The instrument which relatively is most useful is that of Ullmann of Zürich.

It cannot be sufficiently insisted upon that in order to establish the diagnosis exactly in the upper extremity, and particularly in the hand, besides a clear conception regarding the location and physiological action of the muscles, there must be a knowledge of their innervation. We observe, especially, how the ulnar and median are distributed in the small muscles of the hand. The former innervates the hypothenar interossei, the two ulnar lumbricales, and the adductors of the thenar: adductor pollicis, and the deep head of the flexor brevis; the latter, the remaining muscles. In the hand the radial only supplies branches to the skin.

9. Muscles of the Lower Extremity.—(*a*) Muscles from the pelvis to the thigh.

M. ileo-psoas (N. crural is from the lumbar plexus) flexes the hip-joint; it is assisted (and in the sense of pure flexion) by the action of tensor fasciæ latæ (N. gluteus superior from ischiadic plexus). In paralysis of the psoas or of this and the tensor fasciæ it is not possible to flex the thigh either in walking or in bed; paralysis of the tensor fasciæ alone permits the pure psoas action to take place—flexion with rotation outward.

M. gluteus maximus (N. gluteus inferior or plexus ischiadicus) extends the thigh; when the thigh is fixed it brings the pelvis to the horizontal position, and thus the trunk to the vertical (into the upright from the stooping posture, standing upright, etc.). When it is paralyzed there is the peculiar kind of action in rising from the floor described on page 496, with paralysis of the extensors of the trunk.

M. gluteus medius (N. gluteus superior from the plexus ischiadicus), abductor; M. gluteus minimus (same nerve) rotates the thigh inward. The three glutei are the most important supporters of the pelvis.

M. pyriformis (plexus ischiadicus), M. obturator internus (N. ischiadicus), M. gemelli (N. ischiadicus), M. obturator externus (N. obturatorius, plexus lumbalis), M. quadratus femoris (N. ischiadicus) are all, in reality, out-rotators.

M. adductor longus, brevis, magnus, pectineus and gracilis (N. obturatorius, plexus lumbalis) are, for the most part, adductors, at the same time partly flexors. The effect of their paralysis is clear.

(*b*) Muscles from the pelvis and the femur to the leg:

M. quadriceps (N. cruralis) extends the leg; its long head, the rectus, arises from the pelvis (anterior inferior spine), and hence acts with more power when the thigh is in a position of extension with reference

to the pelvis. In paresis of the quadriceps the leg (or possibly both legs) in walking is frequently set forward, flexed more markedly at the knee-joint (the leg during the forward movement of the limb hangs vertically down); and this is true also when it is set down quickly, so that there is a sort of snapping of the knee-joint into the position of extension. The examination is best made by endeavoring to flex the limb when it is actively extended.

M. sartorius (N. cruralis) is probably chiefly an inward rotator of the flexed leg.

Mm. biceps femoris, semitendinosus and semimembranosus (N. ischiad.) flex the knee-joint; the first rotates the flexed leg outward, the second inward. If the limb is powerfully extended by the quadriceps, then these flexors, as well as the gluteus maximus, act: they place the pelvis in the horizontal position (important in walking).

(*c*) Muscles from the leg (or the condyles of the femur) to the foot and toes:

M. gastrocnemius, soleus, plantaris (N. tibial.) are extensors; that is, are plantar flexors of the foot, and, at the same time, adductors of the extended foot.

Mm. peroneus longus and brevis (N. peroneus) are extensors (chiefly the first) and adductors of the foot, lift the outer border of the foot. In paralysis of the peronei muscles (by "peroneus-paralysis" we mean paralysis of the whole peroneus nerve: see below, under M. tibialis anticus): the foot in extension, as well as flexion, stands in the position of adduction and the outer border of the foot is deeper; the foot becomes flat. It is not easy to test the activity of the peronei: we must first show the patient the movements of abducting and lifting the outer border of the foot by passive movements, and then have him repeat them; besides, we have the patient extend the foot: in paralysis of the peroneus longus decided adduction then takes place.

M. tibialis anticus (N. peroneus) flexes—that is, dorsally flexes and adducts—the foot; Mm. extensor digitorum communis and extensor hallucis longus (N. peron.) flex and adduct the foot and extend the toes. Paralysis of the dorsal flexors causes the point of the foot to drop when the foot is lifted from the floor. If the peronei are likewise paralyzed (peroneal paralysis; that is, paralysis of the peroneus nerve), then the foot is lax at the ankle-joint; the point of the foot hangs down, with inclination to adduction. In walking we observe that the foot, as it is raised from the floor, makes a peculiar shuffling motion inward, and it is set down in a fumbling manner. Persons with unilateral, isolated peroneal paralysis are always inclined to take a longer step with the disabled limb in order to obtain the sweeping motion required for the awkward placing of the foot upon the floor.

M. tibialis posticus (N. tibial.) is an adductor.

Mm. flexor digitorum communis longus and brevis (N. tibial.) are flexors of the middle and terminal phalanges of the toes; Mm. interossei externi + interni (N. tib.) are flexors of the first, extensors of the middle and terminal phalanges—interossei externi. [The outer three muscles are abductors of the second, third, and fourth toes respectively, while the first is an adductor of the second toe and assists the plantar interossei.]

Paralysis of the interossei causes a peculiar kind of claw-position exactly analogous to that of the fingers.[1]

M. extensor hallucis longus (N. peron.) extends the first phalanx of the great toe; Mm. adductor, flexor brevis, abductor hallucis (N. tib.) act essentially in accordance with their names: they produce simultaneously flexion of the first and extension of the terminal phalanx. Paralysis of the flexor of the great toe hinders one in walking, but especially in springing.

DISTURBANCES OF SPEECH (LALOPATHY).

I. Dysarthria and Anarthria.

By these expressions we understand those disturbances of speech in which we see it altered in the same way as the activity of a joint is distributed as to its motility—by paresis, paralysis, trembling, spasm, and even ataxia of the vocal muscles.

Unilateral paralysis of the muscles of speech occurs in unilateral affections of the pyramidal tract above the medulla oblongata or of the cortical center of the motor-speech muscles; likewise in peripheral paralysis of the hypoglossus and facial nerves. At first the speech is decidedly disturbed; if these affections continue, there occurs a considerable improvement in the speech, as if it were reacquired by practice. Bilateral paralyses generally occur from the bulbus of the oblongata (bulbar paralysis), and are then, if due to diseases of the motor nuclei, degenerative-atrophic. It is rare to have bilateral speech-paralysis from bilateral cortical or pyramidal lesion (pseudo-bulbar paralysis). We also rarely have a bilateral paralysis of peripheral origin of the hypoglossus or facial nerve.

For the muscles that produce speech and their innervation see pages 494 and 495. Depending upon which muscles are paralyzed, the disturbance of speech may vary with different letters, as mentioned at the above-named place. We recognize slight anarthritic disturbances of speech by requiring the patient to pronounce difficult words quickly, especially such as contain many consonants. The slightest degree of bulbar dysarthria, on the other hand, is sometimes only revealed by the very decided tendency to weariness on the part of the muscles of speech; if the patient begins to recite the alphabet or to count continuously, he starts off very well, but soon his words become indistinct.

Simultaneously with this disturbance of speech, the voice, from paralysis of the palate, is often nasal (or also a kind of "clod-voice"), or the voice has a monotone, or it is inclined to change to a falsetto. Regarding swallowing, see page 494.

Scanning speech: sounding like the speech of a rider of a horse that is trotting; there are sharp changes of rhythm, unnatural pauses, sudden, "explosive," and then, again, snapping pronunciation of words. It is particularly characteristic of multiple sclerosis.

Hysterical dumbness is a complete loss of speech, and generally also of the voice, which occurs suddenly, and generally after an attack of hysteria, which lasts anywhere from days to years and may suddenly disappear. The mobility of the tongue is normal.

[1] See p. 499.

II. Aphasic Disturbances, Disturbance of Graphic Communication (of Mimicking, of Singing).

In order to understand these conditions it is necessary to make some fundamental explanations regarding the processes of acquisition and use of speech, of writing, and also of mimicking.

Speech and writing are both means by which we give sensible expression to our conceptions and thoughts, which can be grasped by others through the ear and eye. Again, by the conception of the word spoken and written by others we partake of their conceptions and thoughts. Speech and writing have come into use among men by agreement for the purpose of making their thoughts understood. There are many different languages, and every spoken or written language must be learned by practice. Now just as the impressions which the spoken words make upon the ear and which writing makes upon the eyes are fine and complicated, so too the muscles of speech by which we utter words and of the hand and arm by which we write words are fine and complicated. Therefore we *acquire* the ability to understand the speech and writing of others, and in turn to utter speech and to form the characters, by accumulating in the cortex of our brain a store of images of these signs.

Let us proceed from *speech*, because this is first learned. The child acquires it, in the first place, by collecting acoustic images of remembrance, images of the sound of words collected from the letters, words and sentences spoken by others. It then itself proceeds to produce these sounds by trying to imitate them; it regulates them by the ear, it improves, and finally acquires the difficult and fine control of the muscles for speech required for the production of sound. This can only be done by remembering the complicated motions necessary for the enunciation of speech—that is, by preserving the recollection of them in the brain.

Increase in the development of a language is marked by an increased number of conceptions, increasing in definiteness and fineness of gradation, first of the concrete and then of the abstract. Next we conceive of the images of the sound of words and the complexes of motion for their production stored at two different parts of the cerebral cortex; but perception, the understanding, rules over both, although not concentrated at a circumscribed spot, but as the result of the co-operation of innumerable tracts and cells. The images of the sound of words heard produce conceptions, the conceptions in turn produce again the images of the sound of words, for we are able to cause a word to sound *internally*, and the conceptions produce the complexes of word-movements: we speak audibly a word which we think of. But in this way it is conceivable that an image of the sound of a word, coming through the ear, directly excites a complex of word motion without the stimulation necessarily passing through the understanding, for we are able to speak words that have been heard without thinking about them—that is, to transfer an image of the sound of a word directly into the spoken word, as we can also repeat the words of a foreign language which we have not understood.

We now come to *writing*. There are stored in the brain the

images of the recollected written signs of words and sentences which we have read, and likewise complexes of motion for the right upper extremity for the production of the images of writing. The image of writing which we have read touches the corresponding conception, the conception in turn produces a complex of writing motion: we write down a word we have thought of; but the conception may produce inwardly an image of the written word: close the eyes and try to see some word thought of. Lastly, the seen image of writing may produce the complex of writing motion without the aid of conception: we are able to copy "thoughtlessly," "mechanically."

And now let any one think of the images of the sound of words stored in the brain and the complexes for producing writing in manifold connections, associations, which associations, however, previously exist, as they enable us to learn to read and write. All these complexes or images of recollection, however, are directly connected with the faculty of conception, at any rate most of them, and possibly all.

In this way it comes about that each of these images of recollection may be produced, innervated from different sides. And indeed there may be produced:

1. *The representations of the sound of words:* these come from the periphery through the sense of hearing. If we hear the mother tongue (or any other language which we know), from the conception we inwardly pronounce the words.

2. *The written representation:* from the periphery—that is, from the organ of sight if we read in a known language; and from the conception if we inwardly represent to ourselves the printed or written word.

3. *The complex motions of speech:* from the center representing the sound of words by virtue of the imitative instinct—repetition; and from the mental conceptions—independent utterance of thought; from the written image—we read aloud.

4. *The complex motions of writing:* from written words, by virtue of our imitative instinct—copying; from mental conceptions—writing out the thought; from the sound-image—we write under dictation.

The conceptions of musical notes seem to co-ordinate those of word-sounds, while the complex motions for producing speech and those which produce music (melody and rhythm)—that is, for singing—are co-ordinated with the larynx and mouth. The conceptions of musical sounds are intimately connected with those of word-sounds, and the complex motions required in singing are connected, through association, with those required in speaking. The intimateness of this association appears very distinctly in the fact that when a melody happens to come to mind we hum the words belonging to it; or, if the words come first, then we hum the melody. Sometimes this humming is a purely automatic act, for both the text and the melody are articulated involuntarily together. But, again, sometimes the internal resounding follows the articulation or act of listening, and from this internal impression the articulation is first produced.

Now to these innervations there belong tracts [of communication].

Those which conduct the impressions to the mind from the periphery we understand very well—the acoustic and optic nerves. Further, there must exist very manifold combinations, association tracts, between the conception and the four different centers themselves [mentioned above], but it is very difficult to obtain an exact presentation of these combinations.

For instance, Kussmaul supposes that the tract from the center of ideas to that for the complex motions of speech goes through the portion which takes note of the sound of the word; hence he assumes no direct innervation of the center of the complex motions of speech from that of ideas, though others think there is. Likewise, there is a dispute whether there is a direct communication from the written representation, or whether there is a communication with the center for the complex motion of writing, etc. only through another center. We will only bring forward one instance, for the sake of illustration. The following acts, done without understanding by persons in health as well as by sick persons—repeating, reading aloud, copying, or writing from dictation—make it plausible that direct communication exists between the sensory and motor-centers, which therefore do not go through the center for ideas. But there is no doubt that in regard to this there are very considerable individual differences, particularly dependent upon the degree of cultivation and the intelligence.

Of course we also understand the tracts which peripherally lead from the "motor-speech and writing-centers" in general run with the pyramidal tract to the motor nuclei of the bulb, or to the cervical enlargement of the spinal cord, and from there to the respective motor nerves. Also the situation of the tract for speech within the pyramidal tract is partly known.[1]

Likewise the situation of the center for the perception of speech and writing, as well as for the centers of the complexes of motion for producing sound, is known. Two of these, speech- and hearing-centers, are unilateral, and are situated only in the left hemisphere; the center for the perception of the images of writing, on the other hand, possibly exists on both sides.

The center for the motor complexes of words, the motor-speech center, is located in the third left frontal convolution (Broca); the center for the images of acoustic recollection, the sound-image center, is in the left temporal lobe in the first temporal convolution (Wernicke). The right hemisphere has nothing to do with language, except in left-handed people, in whom sometimes this center is located in the right instead of in the left hemisphere.

The center for images of optical recollection (images of writing) is to be found in the optical cortical field of the occipital lobe, either on both sides or, more probably, only on the left. A center for the complexes of writing motion formerly was assumed to be in the second left frontal convolution; but it seems that such a secluded center does not exist.

Observe that the centers all lie within the respective motor and sensory cortical fields.

These very different qualities, acquired by practice, may each singly

[1] See p. 418.

or several together be lost by reason of local disease of the respective parts of the brain. When the organ of hearing remains perfectly intact the innervation from the periphery of the conception of the sound of words—that is, the ability to understand the words of one's native tongue—may be lost: there is *word-deafness* ["inability to understand spoken words, although they are heard as sounds, while printed or written words are understood"—Billings], loss of intellectual perception of sounds. Even when the muscles of speech are perfectly normal the ability to employ language to express one's ideas, through the innervation which results in the complex motions necessary to make use of the appropriate word in the native language, may be lost—motor or ataxic aphasia (or, as Kussmaul designates it, "the purest form of ataxia aphasia"). The arm may be in perfect condition, and yet we may not be able to write; or the eyes may be intact, and yet we cannot read—*agraphia*, *alexia*. But since the different capacities under consideration—the understanding and formation of words, the understanding and production of writing—are in a very manifold way connected with each other, these disturbances almost never occur singly, but as a complex of disturbances.

The expressions "*acoustic amnesia*" for word-deafness, "*visual amnesia*" for loss of intellectual perception of sounds, seem to us to be very useful, more so than the German designations formed upon a different principle. The only objection is that these expressions may be confounded with the idea of amnesia discussed later on (page 508).

The study of these things has proceeded from the observation of the disturbances of speech in the narrowest sense; that is, of speaking (Boilliaud, M. Dax, Broca). For this reason, and because all disturbances that come under consideration apply to speech in the broader sense (spoken and written speech, with reference to its comprehension and production), we class together, not at all incorrectly, all the conditions under consideration by the designation of *aphasia*, *aphasic disturbances*.

We mention now in the following details only those two manifestations which may be most sharply distinguished, while for all the details we refer to the special works.[1]

1. **Word-deafness** (Kussmaul), **Sensory Aphasia** (Wernicke). (The two conditions are not wholly identical.) Word-deafness is caused by a local disease (loss of blood, softening, tumor, abscess, or trauma) in the region of the center for images of sound—the left first temporal convolution. The patient hears every word, but it sounds to him as any healthy person hears a word that belongs to a language which is wholly strange to him. The mother tongue, so far as the understanding of the hearer is concerned, has become a foreign, unknown tongue; also, ability to repeat and to write from dictation is wanting. But, again, sometimes the understanding of writing may fail (*alexia*), and with it the ability to read aloud (see page 511).

But, in opposition to this, the power of volitional writing and to copy written characters, and further, volitional speech, are preserved. Nevertheless, we generally observe a disturbance in this also: very often the wrong words are used, because words that are related by

[1] See also the "schema" of Lichtheim, p. 511.

sense or sound are, from unrestrained association, pronounced and strung together (*paraphasia*), or it may be distinctly noticed that the correct words are employed, but they are distorted by repetition of syllables, dropping of syllables, transposition of letters or syllables (*literal aphasia, syllable-stumbling*). Moreover, both conditions sometimes have relation with amnesia ("*amnestic aphasia*," see page 508).

2. Atactic aphasia (Broca's *aphemia*, Wernicke's *motor aphasia*) is a symptom of the motor-speech center in the foot of the third left frontal convolution, or of the "speech-tract" from these downward. It consists in this, that the patient is unable to communicate his thoughts by words: he cannot name objects presented to him, although he promptly shows that he recognizes what they are in that he knows how to use them correctly; if told the name of a given object, he cannot repeat its name, though he moves his lips and tongue with the greatest zeal: simultaneous with this there usually is diminution of the power to voluntarily write or to write down what is heard (or write from dictation)—*agraphia*—with the exception of the ability to transcribe from copy, which is usually retained. Thus, in pure cases, there is perfect understanding of what is said and also of what is written, and hence there is neither word-blindness nor word-deafness.

But in one respect the condition of most patients of this character is still somewhat obscure: with reference to the question whether they are able to mentally produce the sound of the word, to conceive of its sound—*i.e.* to mentally sound the word. According to Lichtheim, it is probable that in most or in all such cases this capacity has also been completely lost. But regarding this point it is very difficult to form a positive opinion with respect to these patients.

We cannot refrain from dwelling a little upon this question.[1] We must confess that in these cases we have found that the method employed by Lichtheim, though ingenious, is very uncertain. In order to determine whether the word which designates the given objects is mentally correctly sounded, he requires the patient to tell how many syllables there are in the word or to press the hand as many times as it contains syllables. It is assumed that when an object is presented to a patient there arises in his mind a conception of the sound. What designation does he think of? I hold up a knife before him—does he think "a pocket-knife" or "knife?"—a drinking-glass: "a drinking-glass" or a "glass?"—"pocket-handkerchief" or a "handkerchief?" I admit that there are substances about which there is no doubt, but one would be easily inclined to hold that the number of syllables was wrong, and yet the patient thought he had understood and had spoken correctly.

There are *slighter forms of atactic aphasia*, which only show a slight defect in the command of language: single words are omitted or single words are defectively pronounced: "doltor," "dolner," for doctor; "lit," for lip; I am "benter," for better, etc.; that is, there is a *literal ataxia, syllable-stumbling*. But often the patient dwells upon only a few words or only one, or even a single syllable, which is constantly employed for everything, as was the case with a patient reported by

[1] For further regarding the examination of patients with aphasia, see below.

Strümpell, and whom we have watched for years, who could only say "bibi, bi-bi-bi-bi-bi." We also have cases of *paraphasia.*

An atactic-aphasic patient who, before becoming affected, was a good singer, may lose the power of singing as well as of speaking, and yet the "ear" may be retained: he hears when he himself or some one else sings a false note. But though the speech may be lost, he may still retain the power to sing the melody of a song, and then it may happen that with the melody he may automatically articulate the words to which it belongs, although he cannot articulate them without the melody.[1]

These conditions, relatively simple and easily understood, are however generally complicated by accompanying anomalies of reading and writing. The phenomena of disease thus become very manifold and often difficult to explain, the more so because, regarding many of the associative relations between the individual functions, we still have only uncertain conceptions, and, moreover, because the peculiarities of individual persons play a large rôle. To mention only a single example: it is in general assumed that the motor-writing center is innervated, not directly from the conception, but by this only through the motor-speech center.[2] This opinion is based upon the circumstance that motor aphasia is frequently combined with loss of the ability to write at will. However, it seems to be undubitable that many persons innervate the writing-center through the sound-image center, that even persons accustomed to write directly innervate the writing-center from the conception. Disturbance of the motor-speech center in such persons will not be followed by agraphia. This one example suffices to show how great difficulties are here presented.

Apart from the destruction of centers, there also are complexes of symptoms which can only be explained by interruptions of tracts of associations between the centers. A study of the scheme of Lichtheim, given further below, will throw light upon this point. Let it only be mentioned here in particular that paraphasia and paragraphia seem to point to an interruption of the tract between the sound-image center and the motor-speech center.

There is another disturbance which plays an important part in all forms of aphasia, and which presents a special group of symptoms: it is *amnesia, amnestic aphasia.* It has no definite localization.

The patient presents a perfect picture of a person who is endeavoring to speak a foreign language which he only slightly or very imperfectly understands. An object is held up before him: he is not able to name it; he repeats it without understanding it, or he remarks: "Yes, certainly, that is the word;" or he hits upon the correct word through association, as upon the number of fingers held up before him, by counting, "One, two, three, four—correct: four." This amnesis manifests itself only with reference to certain kinds of words, as for proper names, or chiefly for those representing the most concrete ideas (Kussmaul).

Amnesia can be mixed with the different forms of aphasia; the

[1] In connection with this, the reader is referred to p. 504 for what was said regarding the connection between the complex motions of speaking and singing.

[2] Compare Lichtheim's scheme, p. 511.

former may be very indistinct—even for a time or continuously may predominate over the aphasia; but it also occurs in all possible conditions that do not at all belong here—senile dementia, disease of the brain of all kinds, in convalescence from any very severe illness, etc. With Lichtheim we do not count these cases as aphasia.

Mode of Procedure in Testing for Aphasic Disturbances.—We look for any possible aphasic symptoms whenever there is disease of the brain, but especially with any patient who has had an attack of apoplexy, and particularly when there is right-sided hemiplegia.

It is evident that the examination of these patients is often interfered with, either because of their mental hebetude—dimness of perception—or the inability to think and the loss of memory which they exhibit. Those patients can only be exactly examined in whom the general effect of the injury has passed off; and the most interesting cases are those where, after the indirect local symptoms[1] have disappeared, an aphasic assemblage of symptoms remains behind as a unilateral disturbance.

In the first place, we ascertain whether there is *amnesia:* if the patient can, we have him count, but further we test him by requiring him to name objects placed before him. If he fails to do this, we give him the name of the object and have him repeat it. If he can do so (either with or without apparent understanding), he is not atactic-aphasic, but amnesic. It is to be remarked that occasionally amnesia may simulate all—atactic aphasia, word-deafness, word-blindness, agraphia.

We now proceed to test for possible *word-deafness* by engaging the patient in conversation, by requiring him to do something, as to touch his nose, or by directing him to take something in his hand—a knife, pocket-handkerchief, etc. We must be careful to avoid making any kind of gesture, also looking in the direction of the object named.

Hereupon we look for signs of *atactic aphasia*, requiring him to speak and to repeat; further, for evidences of paraphasia, literal aphasia. If the patient is atactic-aphasic, then we must always make the effort to discover whether he has the inner sense of words.[2]

After these things we conclude the test by having him read aloud (that is, read with understanding), have him write, compose, write from dictation, copy. With persons who were formerly known to have had a musical ear or could sing, it will be well to inquire whether they retain or have lost these powers, or, especially, what is the relation of the singing of the air and hearing the music to the understanding and speaking of the words that belong to it.

Schemata.—In the examination of an aphasic patient the results are often confusing, and hence it is very advantageous to arrange them for analysis in accordance with a scheme. There are a great number of such schemata; we mention only those given by Kussmaul, Charcot, Wernicke, and Lichtheim. In using them, it should be clearly remembered that they furnish a graphic picture of the functions and the associations which bind them together, but that they cannot present the facts as they actually exist, which it is impossible in any way to do. Nevertheless, these pictures assist one to understand a given case

[1] See p. 423.

[2] See p. 507.

by furnishing a frame-work for recollection, being as it were a skeleton.

An excellent graphic scheme is that of Charcot, which is given below. It gives the four centers in their position in the brain, arbitrarily assuming a secluded writing-center. If we examine the connections of these centers with each other and the double arrows drawn upon them all throughout, we notice that Charcot assumes that each center may be innervated from every one of the others. That, however, ought not to be thought to occur in every single individual; on the contrary, in some these connections are more developed than in others

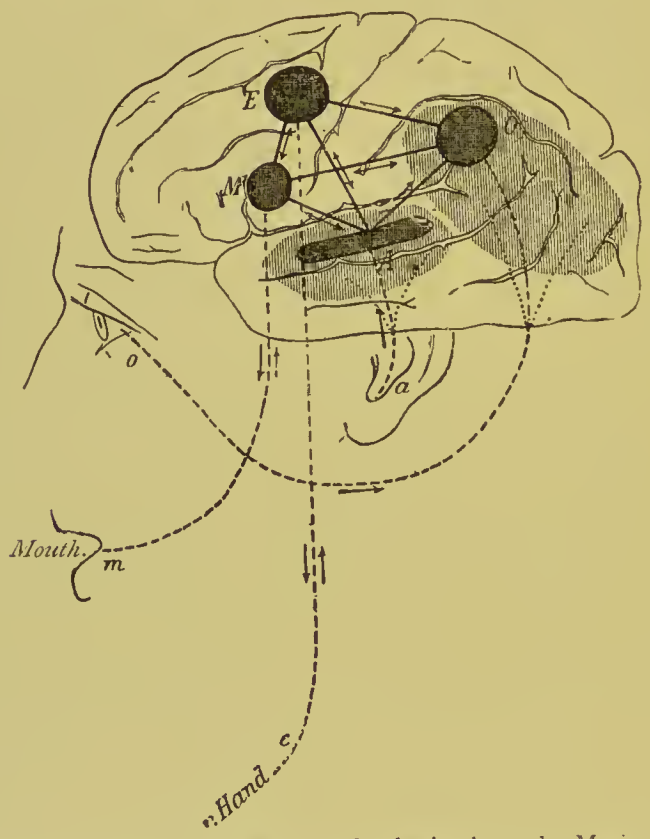


FIG. 179.—Charcot's diagram of aphasia, drawn by Marie.

The designations are the same as in Lichtheim's diagram. The centers are represented as being in those centers of the cortex where they are to be looked for; the light hatching around *A* and *O* indicates the general acoustic and optical field in the cortex. [Notice the double arrows upon all connecting lines between *A*, *O*, *E*, *M*. Also notice the arrows pointing centripetally toward *Mm*. and *Ee*, where the stimulation going to *M* and *E* causes the motions of speech and writing. In our opinion there is to be added the center for ideas, which should have a twofold connection with *A*, *O*, *E*, *M*.]

[and those most fully developed in some cases differ from those most developed in others], as Charcot himself distinguishes a "visual" and an "acoustic" origin of thought. By this he means to say that one person is more influenced in the formation of perceptions by the images of writing, while another is affected most by the images of sound, and this especially applies to the complex of motion for words.

A center of perception [or conceptions] is not shown in Charcot's scheme, as in fact there is no such center. But at any rate, we must imagine such a center and think of it as connected by a twofold means of conduction with all four centers.

We notice that from the mouth and hand centripetal arrows go to the respective motor-centers. They signify the conscious and unconscious sensations which go to these centers from the innervated muscles themselves and from the motions which they produce, and through which we continuously superintend the correctness of our coördination.

Lichtheim's is a purely graphic *schema.* We present it here, and add the combination of possible disturbances and their signs which Lichtheim has arranged with reference to this scheme. Any one who has read attentively the preceding statements will be very well able to

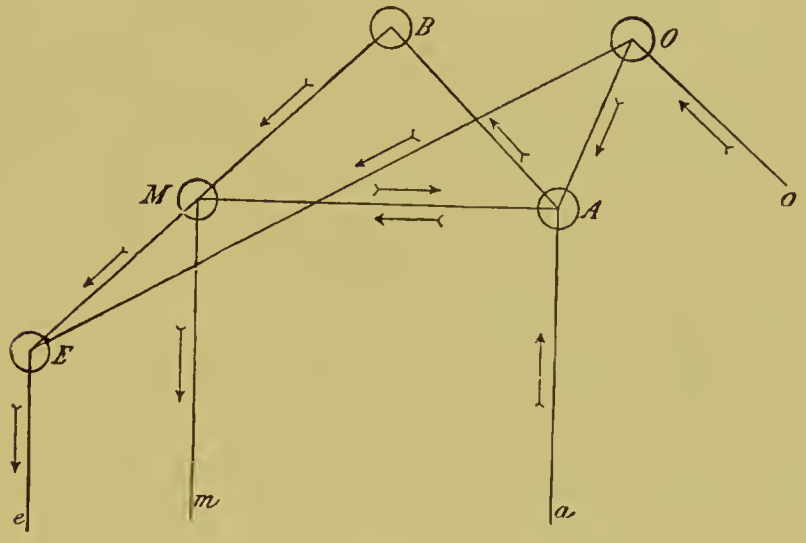


FIG. 180.—Lichtheim's diagram of aphasia.

A, center for conception of the formation of sound (*aA*, conducting tract); *O*, center for conception of written characters (*oO*, conducting tract); *M*, center for the motions of speech (*Mm*, the centrifugal motor tract); *E*, center for the motions of writing (*Ee*, the corresponding motor tract); *B*, center for conception of ideas. The arrows indicate the direction of innervation.

apply the scheme and table of disturbances to a given case of disease. But we cannot here enter upon the considerations which led Lichtheim to produce this scheme. For a more exact study of these points we recommend the classical works of Kussmaul, Charcot and their school, Wernicke, and Lichtheim.

1. Interruption in *M*, the center for the conceptions of motion or the motor-speech center (*atactic aphasia*).

Lost: (*a*) volitional speech;
(*b*) ability to repeat;
(*c*) " to read aloud;
(*d*) " to write volitionally;
(*e*) " to write from dictation (*e* [in the figure], the internal conception of the word-sounds).

Retained: (*f*) understanding of speech;
(*g*) " of writing;
(*h*) ability to write from copy.

2. Interruption in *A*, the center for the conceptions of the sounds of words (*sensory aphasia*).

Lost: (*a*) understanding of speech;
(*b*) " of writing;
(*c*) ability to repeat after one;
(*d*) " to write from dictation;
(*e*) " to read aloud.
Retained: (*f*) " to write volitionally;
(*g*) " to write from copy;
(*h*) " to speak volitionally.

3. Interruption of *MA*.

Intact: (*a*) understanding of speech;
(*b*) " of writing;
(*c*) ability to write from copy.
But there is (*d*) paraphasia;
(*e*) paragraphia (the same disturbance in voluntary writing);
disturbance of the same kind in—
(*f*) repeating after one;
(*g*) reading aloud;
(*h*) writing from dictation.

4. Interruption of *MB*: modification of motor aphasia.

Lost: (*a*) power of voluntary speech;
(*b*) " " writing,
—as in atactic aphasia.
But intact are not only
(*c*) understanding of speech;
(*d*) " of writing;
(*e*) ability to write from copy;
but besides (*f*) " to repeat what is said;
(*g*) " to write from dictation;
(*h*) " to read aloud.

5. Interruption of *Mm*: modification of motor aphasia.

Lost: All speech; everything else intact.

6. Interruption of *AB*.

Lost: (*a*) understanding of speech;
(*b*) " of writing.
Disturbed: (*c*) volitional speech: paraphasia.
Retained: (*d*) " writing;
(*e*) ability to repeat what is said;
(*f*) " to read aloud;
(*g*) " to write from dictation.

7. Interruption of *Aa*.

Lost: (*a*) understanding of speech;
(*b*) ability to repeat what is said;
(*c*) " to write from dictation.
Retained: (*d*) power of volitional speech;
(*e*) " " writing;
(*f*) understanding of writing;
(*g*) ability to read aloud;
(*h*) " to write from copy.

As an addendum we add here a few remarks upon the diagnostic value of the character of the writing:

(*a*) Writing is the expression of thought, and for this reason it is a very fine test for recognizing psychical disturbances of all kinds.[1]

(*b*) As was mentioned above, *agraphia* belongs to the group of aphasic symptoms, and, in fact, it occurs in those forms which are completely analogous to disturbances of speech in the narrow sense: as *total* or *partial agraphia*, as *paragraphia* or *literal agraphia*. Likewise, it was previously stated that a sharp distinction was to be made between volitional writing, writing from dictation, and copying. Also, the loss of the capacity to form strictly grammatical sentences, to make a correct sequence of words (*agrammatismus*, *akataphasia*), shows itself in the writing, also, or still better than, in speaking.

(*c*) *Motor disturbance of the right upper extremity* manifests itself in many cases in a very characteristic way in the handwriting: the different kinds of trembling, ataxia, the different varieties of writer's cramp. It is also worthy of note that patients with paralysis agitans very frequently write naturally, because, as is well known, their trembling ceases when making intentional motions.

The value of the handwriting for diagnosis here consists chiefly in the fact that we may recognize early slight disturbances (the contour wavy): ataxia manifested by the strokes going beyond bounds, especially by the imperfections of the large letters.

In paralytic dementia the disturbance of writing as well as of speech is extremely well-marked. This shows the psychical disturbances: delirium with exaltation or dementia; there is agrammatismus, akataphasia, paragraphia, especially literal paragraphia in an extraordinarily high degree; lastly, there may be motor disturbances of the upper extremities: trembling, ataxia.

SENSE ORGANS.

The Eye.—In considering the relations of the diseases of the eye to internal diseases, those in connection with the diseases of the nervous system are of very much the greatest importance.

We find the eyes, or the function of sight, sympathetically affected in a great variety of ways in diseases of the nervous system. We observe disturbances which exhibit the more or less direct results of disease of the nerves or of the brain. They are—paralyses, less frequently spasms, of the outer and inner muscles of the eye; disturbances of the different qualities of vision itself, from lesion of the sensory tract at any point from the optic nerve to the cortex; neuritis optica (choked disk), which, on the other hand, may itself cause disturbance of vision. Other conditions, which are co-ordinate to the diseases in which they occur, oppose these conditions. They are of extremely varied character. We mention as examples atrophy of the optic nerve in tabes dorsalis, multiple sclerosis, embolus of the central artery of the retina with simultaneous embolus of the fossa of Sylvius, syphilitic iritis or retinitis in syphilis of the brain.

Likewise, the disturbances of the apparatus of vision, occurring with

[1] See the text-books upon Psychiatria.

any other internal diseases, may be either co-ordinated conditions or sequent phenomena of those diseases. Of the former category we name as examples choroidal tuberculosis in acute miliary tuberculosis, retinal hemorrhage in general hemorrhagic diathesis (sepsis, pernicious anemia), the various manifestations of syphilis, etc. As a sequent phenomenon we have embolus of the retinal artery in endocarditis aortæ or mitralis, possibly cataract with diabetes mellitus, etc.

We give these instances in order to show in how great a variety of ways the disturbances of vision may occur as symptoms of other diseases. In what follows we cannot classify the subject matter according to the points of view mentioned above. We rather proceed in accordance with the course of an examination of the eye.

1. Movements of the Eye.—As is well known, these take place, in part at least, in a very complicated way, by the co-ordinate action of the muscles of the eye. Paralysis or spasm of the outer muscles of the eye causes a defective motion of the eye and disturbs its binocular motion, which we designate as *strabismus*. If the strabismus is due to spasm, it is present in all positions of the eye; but if dependent upon paralysis, then it has a different relation. In slight paralysis (paresis) of a muscle strabismus only occurs when a motion of the eye is made which is in a considerable degree dependent upon the co-operation of the muscle paralyzed; on the other hand, in more marked paralysis strabismus may be almost always present. It is only absent when the eyes are brought into a position which corresponds with an especially marked relaxation of the paralyzed muscle. In long-continued paralysis of one or more muscles of the eye contracture of the antagonizing muscles also takes place, in consequence of which condition strabismus is always or almost always present. *Lateral strabismus* is designated as *divergent* or *convergent*, according as there is divergence or abnormal convergence of the axis of vision.

The direct result of strabismus is double-vision, or *diplopia*. This results from the fact that in fixing an object whose image only falls upon the macula of the normal eye in the one whose muscle or muscles are paralyzed, it falls to one side of the macula, and at varying distances from it, according to the degree of the strabismus and the distance of the fixed object from the eye. In consequence of the double image the determination of the position of an object in space, and with it the judgment of the patient with reference to his own position, is disturbed. Hence, primarily there is difficulty in taking hold of objects and in walking; there is dizziness (*vertigo of the eye*), and this is most marked when there is diplopia in looking downward (paralysis of a rectus inferior, of an obliquus superior). But after long-continued strabismus double vision disappears, for the patient learns to voluntarily shut out the abnormal eye.

If it happens to be a case where there is paralysis or spasm of the muscles of both eyes which effect the conjugate motions of the eyes (as the rectus internus of the right and the rectus externus of the left eye), then we speak of *conjugate paralysis of the muscles of the eyes* or *conjugate spasm of these muscles;* for the position of the eyeball we employ the designation *conjugate deviation.*

Paralysis of all or of almost all of the muscles of an eye results in

protrusion of the ball—*exophthalmos paralyticus.* Marked or total paralysis of the oculomotorius produces, besides the paralysis of the eye,[1] also *ptosis* (depression of the upper lid), *dilatation of the pupil, paralysis of accommodation* (paralysis of the levator palp. sup., of the sphincter of the iris, of the muscle of accommodation).

Deviation of the eye in which the paralysis or spasm is located is termed the primary deviation. In cases of paralysis there occurs in the normal eye a so-called secondary deviation if we have the patient cover the normal eye and then have him look with it at an object which has been fixed by the diseased one.[2]

We employ our own individual judgment in determining a paralysis of the muscles of the eye by controlling the position of the eye of the patient while he is looking at a distant object and from the accommodation; also, especially by motions of the ball sideways, upward, and downward; moreover, we test the patient by having him look at objects in different directions, and then question him as to double vision and in what relation the objects stand to one another.

Mode of Procedure in Determining Double Vision.—We hold up a finger about a meter from the patient, move it up and down, to the right and then to the left, and hold the finger steadily in the position in which the patient has a double image, and then have that position described by him. Then we suddenly close one eye: the patient now declares which image has disappeared. In this way we determine to which eye each one of the double images belongs. Or we take a lighted candle as the object of vision, and alternately cover an eye with a piece of colored glass, and then, of course, the image presented to this eye is colored.[3]

In regard to *the significauce of double vision*, it is first to be stated that when the balls diverge the images are crossed; when there is abnormal convergence, they are on the same side (on the side of the convergence). All the rest follows from what will now be said where we collate the function of individual muscles of the eye and the effects of paralysis.

M. rectus externus (N. abducens) rolls the eye outward. Its paralysis, according to its degree, produces convergent strabismus, which is manifest either in looking straight ahead or in looking only toward the side whose external rectus is affected. The double vision is also upon that side.

M. rectus internus (N. oculomotorius) rolls the eye inward, antagonizing the preceding. When it is paralyzed the in-rotation of the ball is imperfect; there is divergent strabismus, crossed double vision.

M. rectus superior (N. oculomotorius) rolls the eye upward and at the same time a little inward. Rectus superior + obliquus inferior acting together simply roll the ball upward. Paralysis of the rectus superior limits the motion upward; the abnormal eye stares downward and a little outward: there is double vision when looking upward; the image of the paralyzed eye is superimposed upon that of the other.

M. rectus inferior (N. oculomotorius) rolls the ball downward and

[1] See below.

[2] Upon this subject, see works upon the Eye.

[3] For further regarding this subject, see works upon the Eye.

slightly inward; acting with the obliquus superior there is simple downward motion. Paralysis of the rectus inferior: in looking down, the paralyzed eye does not move, but remains directed upward and a little outward; there is double vision, with one image above the other, the lower being that of the abnormal eye.

M. obliquus inferior (N. oculomotorius), if it is paralyzed, in looking upward we have the action of the rectus superior alone: the eye turns somewhat inward. There is double vision upon the same side, one image is above the other or they are side by side, particularly in looking upward.

M. obliquus superior (N. trochlearis), if this is paralyzed, then in looking down the rectus inferior acts alone, turning the eye somewhat inward. There is double vision upon the affected side, especially when looking downward.

Some of these paralyses, if they occur singly, can be easily recognized, and this is especially true of those of the recti. But when several are combined, particularly if the obliqui are involved, there is often the greatest difficulty in making out the exact lesion. A combination which may occur frequently is paralysis of all the muscles supplied by the oculomotorius, with which we may then also have the internal muscles of the eye involving the levator palpebræ superioris. With this *total paralysis of the oculomotorius* the eye is rotated outward (the action of the abducens), there is some exophthalmia, the pupil is dilated and remains so in the presence of light, and there is absence of power of accommodation.

By *nystagmus*, or oscillation of the eyeball, we understand very slight clonic jerking motions of the ball. They are generally conjugate. If they take place in a horizontal direction, then we speak of horizontal nystagmus. It is often most distinct in fixing the eyeball, but particularly with marked rotation movements of the balls sideways or in a vertical direction.

Nystagmus is one of the principal symptoms of multiple sclerosis, but it also occurs in cerebral affections of the greatest variety.

The diagnostic significance of paralysis of the muscles of the eye varies very much: paralysis of several muscles of only one eye always points with considerable probability to the base of the brain or to the orbital fissure and orbit, and this is particularly apt to be the case if, at the same time, there is evidence of a lesion of the optic nerve (disturbance of vision, unilateral choked disk). Progressive paralysis of the muscles of both eyes, sometimes ending in total paralysis of these muscles, indicates a progressive nuclear paralysis (*ophthalmoplegia externa*). It is difficult to estimate the symptomatic value of conjugate deviation with reference to topical diagnosis. When it is present we should always first think of the possibility of a lesion of the posterior corpus quadrigeminum or its neighborhood; but aside from this, conjugate deviation occurs with all kinds of local disease of the brain, especially if recent. Hence, if the deviation is due to paralysis, we infer that the line of vision is toward the same side, but if it is a conjugate spasm, the line of vision is toward the opposite side. In the latter case the head is very often drawn to that side. Paralysis of the oculomotorius of one side and of the extremities of

the opposite side (*crossed paralysis*) points with great certainty to a lesion of the crus cerebri, and this corresponds with paralysis of the third nerve. We can immediately understand this fact if we recollect that the N. oculomotorius dexter passes to the right crus cerebri at its base—that is, it passes alongside of the pyramidal tract belonging to the left side of the body.

2. The Pupils.—Normally, the pupils are circular, and their size in individuals fluctuates within moderate limits. It seems superfluous to give a measurement, for it is best for the physician to develop in himself a standard of measurement. In health the pupils are usually of equal width.

The width of the pupils is determined by the action of two antagonizing muscles—the sphincter pupillæ (oculomotorius nerve) and the dilator pupillæ (sympathetic nerve).

When the pupils are abnormal, the first question to arise is: Are the eyes themselves normal? Then follow these important questions: Is there, or has there been, an iritis or disease of the retina? Are the changes in size and form, as well as mobility of the pupils due to these? For particulars we refer to text-books upon diseases of the eye. In what follows we present an abstract of those conditions which have no relation to disease of the nerves.

The Size of the Pupil.—*Contracted pupil, myosis*, occurs in health during sleep, likewise in old age. Otherwise, myosis is always a sign which must awaken suspicion, and indeed is especially frequent in tabes dorsalis;[1] and also, although more rarely, in progressive paralysis. The degree of illumination also has a marked effect upon the size of the pupil, unless rigid from reflex action.[2] Hence, it is to be examined under moderate illumination. *Dilatation of the pupil, mydriasis*, occurs with marked disturbances of consciousness, severe pain,[2] great anxiety, dyspnea, also with reflex rigidity, with atrophy of the optic nerve, paralysis of the N. oculomotorius; lastly, sometimes with tabes and progressive paralysis.

Effect of Poisons.—Atropin, duboisin, cocain dilate the pupil; eserin, pilocarpin, morphia contract it. These effects upon the pupil are, in connection with other symptoms, employed for diagnosis in cases of poisoning with any of these substances.

Inequality of the pupils sometimes occurs with persons in health, also in people with unequal refraction in the two eyes (with myopia: mydriasis; in hypermetropia: myosis); but otherwise, inequality of the pupils is a suspicious symptom. It occurs in unilateral affections of the brain of all kinds (thus, especially with hematoma of the dura), with unilateral paralysis of the oculomotorius of the opticus (dilatation), and in tabes; besides, it frequently occurs in attacks of migraine, where the pupil on the same side as the headache is either enlarged or contracted, according as there is irritation or paralysis of the sympathetic nerve.

Reflex Changes of the Pupil.—The reflex behavior of the pupil plays a most important rôle in the diagnosis of organic cerebral diseases.

Normally, the pupil contracts if light falls into it, by contraction of

[1] See below, under reflex rigid pupil.

[2] See p. 519.

the sphincter: *light reflex of the pupil.* The tract of this reflex passes through the opticus and the chiasm, where probably a partial decussation occurs, to the optic tract, and enters into relation with the anterior corpus quadrigeminum; then it passes centrifugally through the oculomotorius; but the reflex center is still unknown. This reflex contraction of the pupil takes place not only when light falls into the eye being examined, but also if it fall simultaneously into the other: in health the reaction always takes place on the same side as well as crossed. The test is made either in a light room by covering the eye with the hand and then suddenly withdrawing it, or in a room with a dim light by quickly going to the light. In either case the patient must not employ any accommodation, because the accommodation also causes contraction of the pupil; hence, he must look at a distant object. It is best to test each eye singly by alternately closing one. Sometimes there is an indication for testing the crossed ("consensual") reaction: we observe the changes in the pupil of the right eye while we vary the light which enters the left, and *vice versâ.* In old age the reaction of light is physiologically slow.

Pain, as painful irritation of the skin, from pinching or from use of faradic brush, dilates the pupil through the action of the dilator. The reaction is slower and less marked than from light.

Absence of reaction upon exposure to light and when there is pain is called "*reflex rigid pupil.*" For the former anomaly we have recently adopted the shorter, clear expression of *light rigidity of the pupils.* The historical designation, "Robertson's phenomenon," fortunately has not become naturalized.

Unfortunately, the expression reflex rigidity of pupil frequently occurs in literature when light rigidity is meant.

Light rigidity is an extremely important early symptom of tabes and progressive paralysis. In the former disease it is also quite regularly combined with pain rigidity, and also in about half of the cases of myosis. Sometimes, instead of complete rigidity, we notice a slow reaction which, indeed, can only be correctly estimated by the practised eye. Light rigidity or slow reaction, according to former statistics of Erb, occurs in 85 per cent. of all cases of tabes, which number on the whole is probably correct. Light rigidity is not so frequent in paralysis, but here it occasionally occurs a long time, even many years, before the appearance of psychical symptoms (Moeli, Siemerling). It may be combined with myosis, mydriasis, or inequality of pupils; but any of these anomalies may be present without light rigidity.

Further, reaction to light is often absent in syphilis of the central nervous system, when there is lesion of the reflex arch (Uhthoff), although usually it is not clinically demonstrable. Lastly, it occurs as an individual symptom in all possible diseases of the brain (tumors, multiple sclerosis, injuries of the head, etc.), as well as in disease of the base and orbit, whenever they injure the opticus or the oculomotorius, as is usually the case in diseases of the second and third nerve.

In cortical blindness reaction to light is preserved.

The examination of *consensual* or *crossed reaction*[1] may sometimes

[1] See above.

be very nicely used in doubtful cases for determining in which part of the reflex arch the lesion which produces the light rigidity is located. For instance, if the right pupil reacts normally but the left does not, we then test to see whether the left pupil contracts when light is thrown on the right eye. If it does, its motor tract must be intact, and the lesion must be in its centripetal tract. If it does not react, while simultaneously the right pupil contracts when the left eye is illuminated, the lesion must be in the left oculomotorius tract.

So in unilateral atrophy of the opticus, reaction of the diseased eye during illumination of the sound one is preserved; but during illumination of the diseased eye it is absent in it, as well as in the healthy one. Again, in unilateral complete oculomotorius paralysis in the diseased eye there is no reaction in that side, nor can consensual reaction be obtained. All in all, in *light rigidity of the pupils* it must always be remembered that, without exception, it *points to an existing or impending organic disease;* hence such a disease should be sought for, or, if not found, the patient should be kept under observation.

Light rigidity comes into consideration in making a *differential diagnosis* between tabes and neurasthenia, tabes and multiple neuritis (where it is almost always absent), organic disease of the brain and hysteria; also the question whether we are dealing with the results of an injury to the head or simulation may be decided by light rigidity.

Light Reaction in Hemianopsia.[1]—As in cortical blindness the reaction for light is not affected, so also it will be found preserved if, for example, in cortical hemianopsia[2] a circumscribed illumination of the blind half of the retina be made. This is best done by throwing, with a convex glass, a cone of light from the side of the defective field of view. But, on the other hand, reaction to light is absent in all cases of semi-defective fields of view caused by lesion of the optic nerve, of the chiasm, or of the optic tract up to the respective corpus quadrigeminum. In this case the lesion is just so situated that it brings into co-participation the reflex arch of reaction to light (hemianoptic pupil rigidity to light[2]).

Contraction of the pupil in convergence of the eyes or from accommodation may not take place in paralysis of accommodation (this most frequently after acute diseases, particularly diphtheria), but it may also be retained. This contraction of the pupils during accommodation has its chief diagnostic significance in the fact that it must be avoided when testing for the reaction to light or pain—that is, it is generally retained with reflex rigidity of the pupils.

3. Testing for the Central Sharpness of Vision, the Color-sense, and the Field of Vision.—(*a*) We test the **sharpness of vision** by means of *Snellen's plate* which contains test-letters of different sizes, the number of which is represented by the distance in meters at which a normal eye can read the type. After correcting any possible anomaly of refraction in either eye, we put the plate at a distance at which the test-letter X can be read. The sharpness of vision is expressed by a fraction whose denominator is the number on the plate, and whose numerator is the distance at which it can be read. According to the above, in normal vision the denominator and numerator must be alike; the

[1] Compare Fig. 181 and text accompanying it.

[2] See p. 521.

fraction then is always equal to 1 ($\frac{6}{6}$, $\frac{5}{5}$, etc.); instead of this [the *sharpness of vision* represented by] SV. $= \frac{6}{6}$, in case the eye is diseased we have SV. $= \frac{3}{6}$, etc.[1]

As a matter of course, if we discover a diminution in the sharpness of vision, before we conclude that it is due to disease of the nervous system we must exclude any disease of the refractive apparatus. Here, also, the reader is referred to special works upon the Eye.

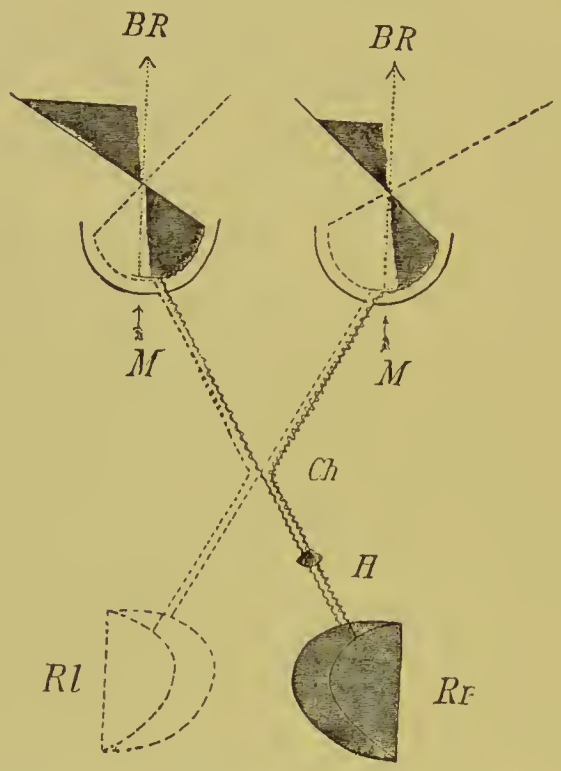


FIG. 181.—Schematic drawing for explaining the relation of the eyes to vision, and representing hemianopsia.

The direction of vision of the two eyes (*BR*) is very nearly parallel (the eyes being fixed upon a distant object). *M*, macula lutea; *Ch*, chiasm; *Rr*, *Rl*, right and left cortical fields of sight (occipital cortex). Notice a kind of semi-decussation in the chiasm, the division of the fibers in the retinæ, and the character of the images as they appear in the cortex. *H*, a local disease behind the chiasm; it causes hemianopsia. The portion of the field of vision which disappears, and the cortical field which does not perceive the object, are hatched. The corresponding tracts are represented by a wavy line.

(*b*) **Testing the Field of Vision, FV., the "Peripheral Sight."**—The most exact way to do this is to employ a perimeter. A substitute for this expensive instrument, which can be recommended to one who is not a specialist, is the field-of-vision chart, which has six straight lines intersecting each other at a point making angles of 45 degrees. Starting from the point of intersection, these lines are divided into centimeters. At the point of intersection a rod of definite length stands perpendicularly to the chart, and sometimes it is screwed into the chart; upon this upright is a hoop, into which the person to be examined places his head. It is used in the same way as a perimeter. The normal size of the field of vision for three or four healthy persons, with a definite length of the upright, is placed upon the chart. (It will be shown that on the outer side the field of vision is endless, because the angle is less than 90

[1] For particulars, see text-books on the Eye.

degrees to the direction of the line of sight—but of this no account is taken.) The pathological result is drawn upon a diagram which represents the chart and the normal field of vision on a smaller scale.

We recognize very decided disturbances by steadily holding a finger about a half meter from and in front of the eye, and then moving the other hand, or a light held by it, in every direction in the field of vision. Of course, in this case, as in all others, we are to test each eye singly. The great difficulty is in having the patient hold the eye fixed immovably.

Concentric Narrowing of the Field of Vision.—This rarely occurs in organic diseases of the brain. It often occurs with multiple sclerosis, usually from atrophy of the optic nerve,[1] more frequently in neuroses; and it is an especially important symptom in hysteria, "traumatic hysteria," but also in "traumatic neurosis," which is closely related to this. With atrophy of the optic nerve there likewise occurs narrowing of the field of vision, which is concentric, more rarely in the form of a sector. *Central scotoma* occurs particularly in alcohol- and tobacco-amblyopia.

The result of semi-decussation of the optic in the chiasm is the peculiar symptom known as *homonymous hemianopsia*—a defect in the field of vision, involving about half of it, upon the same side of the body in both eyes. Fig. 181 explains this condition: a complete interruption of the optic tract or of the path centrally from it, or, lastly, a total destruction of the sight-center in the cortex of the occipital lobe, from which there must result hemianopsia; and, too, the centripetal conduction of the half of the retina corresponding to the side of the lesion will be interrupted, consequently the half of the field of vision opposite the lesion will be defective. Thus, homonymous hemianopsia indicates a lesion which affects the tract of sight between the chiasm and the cortex. Without doubt, this tract also passes through the posterior portion of the posterior crus of the inner capsule, and with equal certainty is in relation with the anterior corpus quadrigeminum of the affected side, for from here also hemianopsia may arise, or, when there is lesion of the corpora quadrigemina of both sides, there is blindness. As one portion of the optic tract, namely, that from the chiasm to the respective corpus quadrigeminum, includes the reflex arch of light reaction, in homonymous hemianopsia which originates from an interruption of the optic tract or in the region of the corpora quadrigemina, we must expect hemianopsic rigidity of pupil.[2] In homonymous hemianopsia this rigidity of pupil is said to be pathognomonic of lesion of the optic tract or anterior corpus quadrigeminum.

Hemianopsia is sometimes made manifest by the patient not noticing when some one comes to his bed from that side; by his not being startled when a light is quickly brought near him from the affected side; or, in writing, he does not see what he has written upon one side of a sheet of paper, etc.

A bilateral dropping out of the nasal half of the retina, with bilateral temporal (hence, not homonymous) hemianopsia, may be caused by a tumor which is situated close in front of or behind the chiasm. In this case the two eyes in some degree may compensate, by mutual action, for the defect, though, of course, very imperfectly—for binocu-

[1] See below.

[2] See above, p. 518.

lar sight is no longer possible. There occur other difficulties whose description does not belong here.

Subjective sensations of vision occur in severe diseases of the eyes of all kinds, but especially in anemia (flimmering), with nervous subjects. Temporary partial amaurosis has great significance: a strong shining, generally pronounced unilateral subjective sensation of light, which, in some of the cases, is markedly present in migraine (*migrène ophthalmique*), sometimes, even during the attack, passing into hemianopsia.

(*c*) **The Color-sense.**—The central perception of color is tested by means of skeins of woollen yarns of as pure colors as it is possible to obtain. The color-sense within the limits of the field of vision—in other words, the size of the field of vision for the individual colors—is ascertained in the same way as that of pure white.[1] It is not without importance.[2]

(*d*) The results of the ophthalmoscopic examination, which are here of interest to us, will be found in the Appendix.

The diagnostic value of the electrical reaction of the retina cannot be determined, hence we pass it over.

Hearing.—Functional Test.—For testing the hearing we use the whispering voice, which, by a healthy person, in a closed room, can be heard at a distance of about twenty-five meters. Each ear is tested separately, by closing first one and then the other by putting the point of the finger into the external meatus. It is to be noted that an ear closed in this manner hears the whispered voice a certain distance, sometimes from one-half to one and a half meters, by conduction of sound through the bones. For this reason it is almost impossible to determine complete unilateral deafness by means of the whispering voice. Another test of hearing is made by ascertaining at what distance the tick of the watch can be heard: a healthy person usually hears it at a distance of one and a half to two meters, though watches differ greatly in respect to the loudness of their tick, and hence a given watch must be previously tested upon a healthy person.

To this also extends the testing of the behavior of the *conductivity of the bones:* a normal person does not at all or only barely hears a watch held near to the closed ear, but hears it distinctly when it is brought in contact with the skull in the neighborhood of the ear. Persons with disease of the outer ear-passage and of the middle ear are in the same condition as those with normal ears when more or less completely closed: at a distance they hear poorly or not at all, but by the conduction of the bones they can hear very well. On the other hand, when the acoustic nerve or its terminations in the tympanic cavity are diseased (nervous deafness), hearing at a distance and through the bones are both alike diminished.

The examination with the ear-mirror is described in the Appendix. Without it it is impossible to make the differential diagnosis of nervous deafness and of affections of the middle ear or of the external ear-passages. The electrical examination of the acoustic nerve (Brenner) has no diagnostic significance.

Apart from the special aural point of view, the determination of a

[1] See above.

[2] See text-books upon Diseases of the Eye.

disease of the ear or of the sense of hearing is of importance for various reasons: (*a*) for recognizing constitutional affections (caries of the petrous bone in scrofula, tuberculosis, middle-ear catarrh in syphilis [1]); (*b*) for recognizing any other local disease of the cranium, or within the cranium (at its base), or of the brain, which injures the acoustic nerve or the central conduction of hearing; lastly (*c*), with reference to further resulting phenomena of a disease of the ear or the petrous bone, if they exist: thrombosis of the sinus, purulent, sometimes, also, tuberculous meningitis, abscess of the brain, and facial paralysis.

It is further to be mentioned that, on the other hand, in a normal condition of the hearing apparatus, a functional disturbance may be caused by a rheumatic facial paralysis near the origin of the nerve: from paralysis of the stapedius muscle, supplied by the facial, and predominant development of the tensor tympani, there may arise a morbid acuteness of hearing, especially for deep tones.

Subjective sensibility of hearing (tingling, ringing, buzzing, roaring, in the ear, etc.) occurs in anemia, nervousness; further, in diseases of this organ of any kind; but, lastly, also in palpable nervous diseases. The latter are then generally affections of the acoustic nerve, as compression or neuritis, or of its terminations in the labyrinth. Subjective auditory sensations, as signs of disease of the acoustic nucleus of the oblongata, or of the auditory tract in its central course, or of the auditory center of the cortex in the temporal lobe, are very rare, if not unreliable. It is very worthy of note that tinnitus aurium may sometimes introduce an attack of migraine, apoplexy, or, as an aura, an epileptic attack.

Tinnitus aurium may occasionally be combined with dizziness (N. vestibularis); this is much the most pronounced in Ménière's disease. Marked ringing in the ears may become the source of psychical disturbance.

It is a notable fact that pain in the ear may sometimes be caused by diseased teeth, just as toothache may be caused by disease of the ear.

In order to make a diagnosis of word-deafness, or of sensory aphasia, it is, of course, necessary, as a preliminary condition, to determine whether the hearing is good.

Lastly, attention must be especially called to the fact that a unilateral disturbance of the hearing may not only have entirely escaped the attention of the patient, but that even bilateral disturbances of hearing may exist for a long time unnoticed by the patient or his associates when it develops gradually. This occurs from the fact that while ordinary conversation can be heard as usual by a person whose hearing is considerably impaired, whispering voices, for instance, can only be heard for four meters or less.

Smell.—*Testing its Function.*—For this purpose we may employ camphor, petroleum, perfumed spirit, and, as disgusting material, asafetida; but not ammonia or acetic acid, because even a very slight amount of the vapor of these substances may irritate the trigeminus. We first test one side and then the other. The examination of the nose with the nasal speculum is described in the Appendix.

Anosmia [loss of the sense of smell] of neuropathic origin is not

[1] See p. 253.

very frequent. It occurs in processes in the anterior cranial fossa and the anterior portion of the brain which lead to compression of the olfactory, as from tumors, meningitis, hydrocephalus; and here it is also due to compression of the olfactory. Unilateral anosmia has been observed as an associated phenomenon of total hemianesthesia in lesion of the posterior portion of the internal capsule: it then exists on the side opposite to that diseased. But in exactly the same way we may have unilateral anosmia with hysterical hemianesthesia. It is rare to have anosmia from lesion of the nerves passing off from the bulb in the ethmoid bone when this bone is fractured.

But it is always to be remembered that the most frequent cause of loss or diminution of the sense of smell is disease of the nasal mucous membrane. It is further to be noticed that in old age anosmia sometimes occurs without any notable pathological cause (atrophy of the olfactory).

In very isolated cases the disturbance is to be referred to paralysis of the trigeminus; that is, to dryness of the nasal mucous membrane due to the paralysis.

Hyperosmia and *osmic paresthesia* (parosmia) occur in hysteria and insanity, and as an aura in genuine epilepsy.

Regarding the significance of the nose as a point of departure in disease within the cranium, compare further on the following page.

Taste.—*Testing its Function.*—We test it for the recognition of salt, sugar, vinegar, and quinin. We also make a test by retaining the same order of succession of all these substances when suitably diluted. Then follows the testing of a circumscribed portion of the tongue, as first one and then the other half of the tongue, then the anterior two-thirds as compared with the posterior one-third, because the former portion is supplied by the chorda, the latter by the glossopharyngeus. For this purpose we wipe the tongue somewhat dry, apply to it a very little of the [test] fluid with a glass rod, remove any surplus and have the tongue simply drawn back, but without any further motion. Although this method is somewhat doubtful, since a portion of the hard and soft palate, which cannot be exactly defined, also possesses the sense of taste, yet it seems practicable, as follows from its positive results in certain cases of facial paralysis. The more exact method of not drawing the tongue back into the mouth after the test substance has been put upon it, thus to eliminate the assistance of the palate, has the disadvantage that then even persons in health can only imperfectly taste.

Ageusis [loss of the sense of taste] on one side of the tongue is observed with total hemianesthesia. Unilateral ageusis of the anterior portion of the tongue occurs also from peripheral chordal paralysis, and this is the case whether it involves injury of the branch of the trigeminus as far as the Gasserian ganglion, or of the second branch from there to the spheno-palatine ganglion, or of the facial between the geniculate ganglion and the point where the chorda is given off, or of the commissural portion between the fifth and seventh nerves, the N. petrosus superf. major. Total ageusis points to hysteria.

Moreover, the fineness of the taste, as well as of smell, varies much with the individual.

DISTURBANCES OF THE VEGETATIVE SYSTEM IN NERVOUS DISEASES.

We must here limit ourselves to a brief enumeration of the most important points.

1. General Phenomena.—The apoplectic habit (short, thick neck, red face, full chest, abundant layer of fat) decidedly predisposes to hemorrhage of the brain, but this also occurs very frequently even in very lean and anemic subjects. In other respects the general habit does not predispose individuals to diseases of the nervous system.

Nutrition.—Nervous diseases affect the nutrition in a great variety of ways, sometimes not at all for a long time, and again very decidedly. It depends chiefly upon the accompanying vegetative disturbances: fever, decubitus,[1] and the various disturbances of individual internal organs to be mentioned.

The tuberculous nature of a local disease of the brain, or of a meningitis may be suspected (aside from possible tuberculosis of the lungs, scrofula, hectic fever) when the nutrition is decidedly poor. The same thing is true with respect to carcinoma.

Fever.—Fever occurs in diseases of the nervous system: (*a*) if the disease itself is of an inflammatory or infectious nature; (*b*) if it causes vegetative disturbances, as decubitus, cystitis, etc., which in turn give rise to fever; (*c*) in many cases where the elevation of the temperature is supposed to be of a neurotic character: in progressive paralysis, in injury of the cervical spinal cord, which is not fatal (here, according to Naunyn and Quincke, the increase in the production of heat rises to 44° C. [= 112° F.]), in tetanus, in severe epileptic attacks.

Diminution of temperature is likewise seen in progressive paralysis and with injuries of the cervical spinal cord.

2. Disturbances of the Respiratory Apparatus.—**Nose.**—Certain affections of the nose (nasal polypi, enlargement of the turbinated bones, chronic catarrh) stand in a peculiar, often causal relation to various neuroses, especially to bronchial asthma, to nervous affections of the heart. The nose, through the ethmoid bone, may be the gate of entrance for meningitis or abscess of the brain; also, it is to be mentioned that the nose comes especially under consideration in the diagnosis of syphilis.

Larynx.—When there is paralysis and anesthesia of the larynx we must investigate its nerves and their centers in the bulb; further, hysteria sometimes comes into consideration.[2] We have a nervous cough from simple nervousness, also in hysteria. "Laryngeal crises" are attacks of nervous cough, which may occur in decidedly varying severity from slight irritative cough to attacks resembling whooping-cough of the severest character. They are produced by irritation of the vagus by tumors of the bronchial glands, or it occurs in tabes and hysteria.

Dyspnea.—See what was said regarding asthma in connection with the nose. It occurs also in uremia, and is sometimes the most prominent symptom in chronic uremia, and in diabetic coma. Lastly, dyspnea is caused by functional and true paralysis of the respiratory

[1] See below. [2] See some additional remarks regarding the larynx in the Appendix.

muscles. With the latter we take into consideration the tracts of the nerves, the nerve centers, especially the respiratory center in the bulb. Dyspnea is caused also by tonic and rapidly recurring clonic spasms of these muscles. In hysteria there is great disturbance of the breathing: extremely rapid superficial, or labored, deep, panting breathing, and temporary fixation of the diaphragm.

Regarding Cheyne-Stokes' phenomenon, see page 81.

The condition of the lungs and the character of the sputum are chiefly regarded from two points of view: the determination of a tuberculosis; and, because a connection between fetid bronchitis, abscess or gangrene of the lungs and emphysema, and purulent meningitis, and abscess of the brain has recently been recognized.

3. Disturbances in the Circulatory Apparatus.—**Heart.**—This has most important relations to hemorrhages and embolic softening of the brain: hypertrophy of the left ventricle favors the occurrence of hemorrhage (contracted kidney) and valvular endocarditis. In case of weak heart, thrombi existing within the heart (the left auricular appendix) may cause emboli. Atheroma of the vessels, likewise, may cause hemorrhage, emboli, and local thrombosis of the vessels of the brain. But often aneurysm of the minute arteries of the brain causes hemorrhages without there being any atheroma of the vessels of the body. Whenever there is loss of consciousness, but especially in every case of apoplexy, and of paralysis which is to be referred to the brain, the heart and vessels are to be most carefully examined.

Palpitation and *pain* (angina pectoris) occur in organic disease of the heart, in simple nervousness (heart neuroses), in hysteria, in Basedow's disease, and in nicotin poisoning. Hence these phenomena may have great diversity of significance.

Much has already been said (page 203, *f*) regarding the *anomalies of frequency of the pulse.* Temporary, seldom continuous, quickening of the pulse occurs in neuroses; but, besides, paralysis of the vagus or the vagus nucleus (neuritis, bulbar paralysis) quickens the pulse, often, also, causes a gallop-rhythm.[1]

The *vaso-motor disturbances* are extremely manifold and interesting, but, according to our present knowledge, are seldom of diagnostic importance. There must be mentioned the unilateral paleness or redness of the head in many cases of migraine (hemicrania, sympathetica spastica and sympathetica paralytica); unilateral paleness in hysterical hemianesthesia. We observe cyanosis, coldness, edema, especially frequent in cerebral, sometimes also in spinal (poliomyelitis acut.) and in peripheral paralyses, and in hysteria. Sensations of heat of the skin in Basedow's disease—perhaps, also, in paralysis agitans—are to be referred to vaso-motor influences. Regarding the secretion of perspiration, see page 32, *f*.

Local asphyxia (cyanosis, coldness) and spontaneous symmetrical gangrene is observed in general neuroses, peripheral neuritis, but also in acute infectious diseases, diabetes, and ergotism.

4. Disturbances of the Digestive Apparatus.—Very much has already been said upon this point, hence reference is made to page 252, *ff*.

[1] See p. 190.

Anesthesia of the pharynx may, exceptionally, be evidence of a palpable disease; it is a much more frequent and important symptom of hysteria.

Increase in the secretion of saliva occurs in psychoses, idiotism, also in bulbar paralysis; in all three cases—in the first from inattention, in the latter from simultaneous paralysis of the lips, tongue, and of the muscles of deglutition—the secretion sometimes runs out of the mouth. But, for the same reason, in bulbar paralysis the secretion escapes from the mouth, although it is not increased in amount. Diminished secretion of saliva is seen chiefly in facial paralysis (secretory fibers in the chorda tympani).

We are also to bear in mind the *nervous dyspepsias*, which may be divided into psychical disturbances, as dyspeptic difficulties with perfectly normal digestion, and nervous disturbances of secretion or of the motor function of the stomach. The diagnosis is to be determined by an examination of the contents of the stomach.

As was previously mentioned, *vomiting* takes place in all kinds of disease of the brain, especially in those that develop rapidly; further, very especially in the course of diseases of the cerebellum. It is also to be mentioned that there is vomiting with migraine and hysteria. *Gastric crises* are attacks of very severe, often widely-radiating cardialgia, associated with vomiting (hyperacidity). They are a peculiarity of tabes, and not infrequently they are for a long time misunderstood. Intestinal crises (attacks of colic) and those involving the rectum (severe tenesmus) are more rare occurrences in tabes.

With a number of nervous disturbances, especially in children, we must think of *intestinal parasites*. They may cause nervous agitation, marked nervousness, attacks like migraine, and spasms. It is not unimportant, although very infrequent, that the tænia solium may infect the subject who has it with cysticercus [cellulosæ]: thus, sometimes, cysticerci may develop in the brain, in the eye.

Habitual constipation is especially frequent in all kinds of diseases of the spinal cord. Marked *retentio alvi* is very often dependent upon weakness or paralysis of the abdominal muscles, perhaps from abdominal pressure.

Incontinentia alvi is partly the result of inattention on the part of idiots, the insane, those who are unconscious; on the other hand, it is evidence of paralysis which only manifests itself either by the fact that the stool cannot be retained long after the first sense of desire, or that only the fluid stool cannot be held back; lastly, that solid as well as fluid stools pass each time. This disturbance may occur from interruption of the reflex arch centripetally from the rectum to the lumbar portion of the spinal cord, and thence again to the sphincter muscles, or by interruption of the tracts, centripetal and centrifugal, between the lumbar cord and the brain (voluntary defecation). Involuntary discharge of the stool likewise takes place, particularly in spinal diseases both of the lumbar cord and of the portion above it. In the latter case the discharge seems to be regulated by the absence of reflex, but without the influence of the will; on the other hand, in destruction of the lumbar cord, the reflex as well as the voluntary influence is annulled: the sphincter is relaxed, the scybala escape as

they are carried down from the intestine. The same thing is also observed in very great prostration [as in typhoid fever].

5. Disturbances of the Urinary Apparatus.—*Oliguria, anuria,* also *polyuria,* may temporarily affect hysterical patients. Polyuria (diabetes insipidus) also glycosuria are observed temporarily or continuously with local diseases of the oblongata, for a very short time in tabes, and when there is considerable increase of the intracranial pressure. On the other hand, in genuine diabetes mellitus there are observed a number of nervous disturbances: neuralgia, neuritis, deep disturbances of the nutrition of the skin and the subcutaneous cellular tissue, and either slowly developing or sudden coma like apoplexy.

Cystitis, from the slightest to the most severe form, is observed when there is difficulty in emptying the bladder,[1] and especially (but not exclusively) after the use of the catheter. It is particularly an important and frequent complication of myelitis transversa and of tabes.

Further particulars regarding the condition of the urine have been given in connection with the urinary apparatus itself.

Involuntary passage of the urine occurs in the insane, with idiots, in the state of unconsciousness, in severe diseases of any sort; further, as a special form of disease in enuresis nocturna.

Retentio et incontinentia urinæ, however, have an especial rôle. With the former, the patient, when urinating, must press or wait a little, when the urine gradually comes in the ordinary way, or else it escapes very slowly in a small stream, or the bladder cannot empty itself at all and the catheter must be used. Incontinence often first manifests itself as under reflex control, but the urine is passed independently of the will, or simultaneously with retention there is an after-trickling, or an escape of the urine while laughing, coughing, or in severe cases, as *ischuria paradoxa:* the bladder is not completely emptied; it sometimes remains always abnormally full, but from time to time some of its contents escape; in the most severe cases the urine trickles continually from the constantly-full bladder. In the latter cases there is complete paralysis of the bladder (generally of the detrusor as well as of the sphincter).

An involuntary passage of urine which is under reflex control requires an intact reflex arch: (*a*) healthy mucous membrane of the bladder; (*b*) sensitive muscle; (*c*) nerves; (*d*) lumbar spinal cord; (*e*) muscles of the bladder—hence it occurs with an intact lumbar cord, but one which is cut off from the brain: myelitis transversa dorsalis, cervicalis, or traumatic and other spinal transverse lesion. We meet with complete paralysis of the bladder chiefly in lesions of the lumbar cord. All kinds of bladder disturbance occur, from the slightest to the most severe, in tabes. Differential diagnosis comes chiefly into consideration from the fact that disturbances of the bladder are absent in multiple neuritis (as against tabes); further, in amyotrophic lateral sclerosis, poliomyelitis (as against myelitis).

We have still to mention the [frequent, but not invariable] involuntary passage of urine in attacks of genuine epilepsy; it is wanting in hystero-epilepsy, and so it is important for differential diagnosis.

Bladder crises (painful tenesmus) are observed in tabes.

[1] See this.

Lastly, it is to be cited that the most varied conditions of irritation of the penis (especially phimosis) may lead to enuresis, pollution, other nervous disturbances of various kinds.

6. Disturbances of the Genital Apparatus.—The various anomalies of the male genital function may be almost entirely (with the exception of azoöspermia and aspermatism) functional and organic, and in the latter case again may rest upon a nervous as well as some other form of disease. From the standpoint of diagnosis of nervous diseases the decline of the genital function is chiefly of importance in tabes, as against chronic multiple neuritis. On the other hand, differential diagnosis from neurasthenia spinalis is often necessary, and it is to be remembered that in the latter disease there may also be long-continued marked functional disturbance of the activity of the sexual function.

Of the female genital apparatus very little needs to be said here. An energetic reaction has taken place against the etiological relation, formerly very strongly claimed, between anatomical disturbances and hysteria, which reaction, in turn, is going too far. In our opinion, there is no doubt that in women diseases of a sexual character may cause hysteria, certainly more than do other conditions which tend to weaken the nervous system.

The so-called *painful ovary* or *ovarian hyperesthesia*, sensitiveness of the hypogastric region, especially on the left side, to pressure upon this spot (which has nothing to do with the ovary) is not unimportant in hysteria and sometimes causes an hysterical spasm; also [pressure] sometimes arrests an existing attack [Charcot]. Similar hysterogenous zones may exist in other regions of the body in hysterical subjects.

7. Disturbances of the Skin.—A number of diseases of the skin, apart from the special province of dermatology, rest upon a neurotic basis, as herpes, sometimes probably also pemphigus; further, the so-called glassy skin; at any rate, each of these may be regarded as a disease of the peripheral nerves. *Herpes zoster* especially, when it involves the intercostal nerves, has a special significance: it has its origin in compression of the spinal cord, in tabes, meningitis spinalis (here probably entirely from the roots of the nerves), in disease of the spinal ganglion, and in peripheral neuritis, in all these cases generally associated with neuralgic pains. But herpes also occurs in the region of any other nerves, as the trigeminus.

Regarding herpes labialis, etc., see under acute general diseases, page 44.

In all diseases of the nervous system we must search carefully for any evidences of *syphilis*, not only upon the skin but also in the other organs which come under consideration.

Regarding local perspiration (see page 34) we sometimes, although rarely, have local anidrosis. Among the laity the loss of perspiration of the feet plays an important part as the supposed cause of a number of diseases, particularly spinal, as tabes; it is probably a consecutive, and in itself an indifferent, phenomenon of this disease.

Hemorrhages of the skin occur spontaneously in hysteria, as curiosities; punctiform ecchymoses may be observed upon the face, chiefly

in the neighborhood of the eyes after epileptic attacks. Here, also, we more frequently have hemorrhages in the conjunctiva. Hemorrhages into the subcutaneous tissues take place after injuries received during an epileptic attack. The significance of hemorrhages into the skin and subcutaneous cellular tissue of the head (especially about the eyes, and of the nose in fracture of the base of the skull), is treated of in the works upon Surgery.

Decubitus is an ulceration of the skin, then of the subcutaneous tissue and sometimes of the deeper tissues, and even of the bone itself. It occurs in dependent portions of the body upon which the patient's weight rests, and particularly where the skin covers bony prominences, as the sacrum, the heels, the scapula. Want of cleanliness, and lying upon the sacrum, especially when there is incontinence of stool and urine, are very marked exciting causes.

1. *Decubitus acutus* (malignus) at first manifests itself as an erythema exudativum, then vesicles are generally formed, whose bases become necrotic, from which the destruction proceeds rapidly both in area and depth. Pressure and filth are marked causes, but pressure alone may produce the ominous exudative erythema, as on the inner sides of the knees when pressed together in cases of adduction contracture, where we once saw an enormous decubitus acutus form in a few days. Decubitus acutus has been seen by Charcot in hemiplegia upon the posterior portion of the paralyzed side two to four days after an attack of apoplexy. We have observed it only in severe diseases of the spinal cord.

2. *Ordinary decubitus* occurs only when the body lies so that pressure is made upon one place and with the concurrence of uncleanness; it may be entirely prevented by proper care. It also begins as an erythema, or in the form of a few pustules, or a cutaneous hemorrhage. It occurs in all organic paralyses, also in any kind of cachexia, if care is not taken to prevent it.

Mal perforant [perforating disease of the foot] is a destruction of the skin and deeper parts of the foot, especially of the heel [sole?]. It occurs in tabes, in progressive paralysis, also in diabetes. Ulcerations of the skin or subcutaneous tissues, also the capsule of the joint and the periosteum have frequently been observed in syringomyelitis of the cervical cord.

Growth of hair is a very notable anomaly dependent upon a neurosis. But these changes have no independent diagnostic significance.

The nails readily become claw-like, angular, and brittle in long-continued severe peripheral paralysis.

8. Bones and Joints.—We observe the arrest of growth of bones after severe central paralysis during the period of childhood, and, likewise, after poliomyelitis acuta it is generally more marked than after encephalitis. Abnormal brittleness of the bones is frequently seen in tabes. In severe syringomyelitis of the cervical cord there are severe trophic disturbances of the bones, as fractures, periostcal inflammations with separation of sequestrum.

Arthropathiæ of all kinds are to be observed in diseases of the nervous system: 1. Organic arthropathia, seldom in recent hemiplegia, occurring more frequently as stiffness of the joints, is easily con-

founded with stiffness and sensibility from contracture. It occurs in old hemiplegias, and is also to be observed as serous effusion with periarticular swelling or as severe deforming arthritis, also causing new formation; both the latter occur in tabes. There also occurs in syringomyelia severe deforming arthritis.

2. Joint neuroses occur as painful, occasionally exacerbating affections of the joint, sometimes with pressure points [tenderness], stiffness, and contracture, the two latter disappearing under narcosis, but without any sign of organic disease.

Under the name of *acromegalia*, Marie has recently described a peculiar disease, which consists in a giant-like enlargement of the feet, hands, nose, inferior maxilla, and certain parts of the skeleton, dependent entirely or chiefly upon hypertrophy of the bones.

Remarks upon the Diagnostic Value of the Symptoms in Nervous Diseases.

In diseases of the nervous system the individual phenomena combine to form complexes of symptoms in so manifold a way (much more than in the diseases of any other organ-system), that the representation of only the most important possible combinations would very much exceed the limits of a brief work upon diagnosis. Moreover, for the introductory study of individual diseases, we must confess that we think the method of special pathology which compactly presents the picture of disease on the lines of etiology, anatomy, and symptoms is far preferable to the introduction of such minutiæ into a text-book upon diagnosis. For this reason we add here only a few general remarks.

In diseases of the nervous system much more than in those of the rest of the organism, the impression stands out distinctly that we in reality have to estimate the phenomena found in a patient in two ways. We must ask ourselves:

(*a*) What are the portions of the nervous system whose disease, judged by their nature and location, has caused or can cause the present phenomena? This proceeds upon our knowledge of the anatomy, physiology, and pathological physiology of the nervous system, which we must acquire as perfectly as possible.

(*b*) Does the picture formed by all the symptoms correspond with any disease with which we are now acquainted? Then comes the further question:

(*c*) What light does the etiology, development, and course of the disease throw upon its nature, and sometimes also upon its location?

The lines of thought designated by (*a*) and (*b*) closely interlock; generally both are employed in a single case. In certain diseases, indeed, we are wholly or almost wholly directed to the latter (*b*), the, so to speak, unscientific lines of thought, particularly in certain *general neuroses* or *functional diseases*. On the other hand, we are fortunately able, in *local diseases* of the brain, of the spinal cord, and of the peripheral nerves, to proceed upon an almost purely anatomico-physiological basis.

In order to make *a diagnosis of the location of a local disease*, besides the special knowledge requisite for such a discrimination, one must have a certain amount of practice in making combinations, of which the ability to keep in mind the topography must form the basis. (Let it be here once more repeated that our preliminary anatomical remarks do not, by any means, contain all that has been positively determined and is interesting to know, but are rather for the purpose of instruction in topographical thinking). We advise the beginner, who wishes to train himself in this department, to begin with the study, for instance, of peripheral facial paralysis, the different combinations of paralysis of cranial nerves at the base of the brain, and then to study the group of symptoms in the cerebral centers.

In order to arive at a conclusion regarding the situation of a local disease it is recommended, as the result of experience, that we should always attempt to trace the different phenomena back to a focus; but it is evident that sometimes there will be several foci. Moreover, the probability that there is only one focus varies with the supposed nature of the disease; thus, for instance, a glioma almost always occurs as a single tumor, while metastatic cerebral abscesses are generally, and thrombotic foci of softening very often, multiple.

In regard to *local diseases of the brain* we are to distinguish between the general phenomena as respects the brain and the local symptoms. We refer to what we have said above on page 423, *f.*

But in all diseases of the nervous system all possible disturbances in the rest of the organism contain diagnostic points, and, for forming a judgment as to the nature of the local trouble, come especially under consideration in local diseases of the brain and spinal cord. We compare what was said upon this point in the chapter on vegetative disturbances; but, especially, we must never fail, in every disease of the brain and spinal cord, to take into consideration the possibility of the syphilitic nature of the disease (when there is the slightest suspicion of syphilis the treatment is to conform to it).

Under the anatomical diseases of the nervous system, in every respect the so-called systemic diseases have a special place. In these conditions the disease in the nervous substance is, with more or less regularity, always concerned only with certain elements, which systematically (in Flechsig's sense, see below) belong together, while other portions, even lying very close to the diseased ones, remain entirely healthy: the disease does not lay hold of the entire region, and thus it stands in sharp distinction from the inflammatory diseases and all new formations. But even though the systemic disease lays hold upon elements of the same function (and indeed always the symmetrical portions of the two sides; and these are generally, although not always, of the same severity upon both sides), it always produces, at least in its main features, a like combination of symptoms. If several systems are affected with disease at the same time, then we speak of the combined system-disease. Amyotrophic lateral sclerosis furnishes the most clear picture of a combined system-disease which may affect the whole cortico-muscular conducting tract from the cortex to the muscles, but always leaves all the rest entirely intact. We advise every one to begin the study of the system-diseases with this remarkable one.

Beside the systematic nerve-trunk degenerations, we also speak of systematic nuclear degenerations, in that we have somewhat modified the idea of the system which was employed by Flechsig only for the bundles of fibers which showed similarity by the point of time when their medullary sheath was formed (and which "appeared to be intercalated between apparatus having objects of equal value"). Hence, and not incorrectly, we designate the disease itself as systematic when it involves "apparatus having objects of equal value."

In conclusion, we make a few further remarks regarding the *differential diagnosis of functional and anatomical diseases of the nervous system.* This differential diagnosis is often so extremely easy that the question does not arise at all, but sometimes it is extremely difficult. The points of departure for the differential diagnosis are arranged in four categories:

1. The first question always is whether the *total picture* entirely corresponds with a local disease, or an anatomical or functional disease. It is to be remarked, however, that hysteria may sometimes exactly simulate a local disease of the brain.

2. There are certain symptoms of palpable disease that are entirely unmistakable. These are: the reaction of degeneration or rapidly developed and very decided atrophy and laxness of the paralyzed muscles;[1] choked disk and reflex rigidity of pupils are also symptoms. Not absolutely certain, although pointing quite decidedly to a palpable disease, are: absence of tendon reflex; in unilateral affections, the unilateral absence of abdominal reflex, and very marked disturbance of the bladder.

3. There is one almost certain sign of functional disease: a sudden return to a perfectly normal condition after long persistence of a diseased condition, or the sudden occurrence of new and different phenomena with the disappearance of those previously existing. There are other signs of hysteria which, in their combination, cannot mislead; these are the *stigmates hysteriques* (Charcot): hysterical hemianesthesia of the skin and organs of sense, concentric limitation of the field of vision, characteristic spasms, sometimes hysterogenic zones.

4. As regards cerebral symptoms, marked development, or on the other hand, the absence, of a disturbance of the sensorium and the intelligence, decides the question. Also, continuous fever and rapid decline of strength points to an anatomical disease.

[1] Compare, further, what was said on p. 456 regarding atrophy in hysterical paralysis.

APPENDIX.

We present here a very brief sketch regarding the examination of the larynx, the nose and the ear with the mirror and the revelations of the ophthalmoscope, so far as they are related to internal diseases, especially to the diseases of the nervous system. Lastly, there follows a review of the life history of those pathogenic bacteria which have any part in the diagnosis of internal diseases.

The examination with the mirror of the nose and ears can only be briefly touched upon, because these pertain chiefly to the diseases of these organs themselves, and are very rarely of significance for recognizing any other diseases. Beside, with reference to the latter view, we have already (pages 66 and 522) referred to the diseases of the nose and ears which do sometimes come under consideration.

1. LARYNGOSCOPY.[1]

Instruments and Sources of Light.—Türck's reflector with a head-band is most frequently recommended for illuminating the throat. As the laryngeal mirror we employ a round mirror, with a diameter of 20 to 25 mm., fixed to a staff at an angle of 120 to 125 degrees. The staff is fixed to a handle or it is screwed into a handle prepared for it.

For a source of light we may employ any sufficiently powerful oil- or gas-lamp. The lamp is placed close to the head of the person to be examined, so that the light from the reflector is thrown at the smallest possible angle into the throat of the person being examined. If it can be had, sunlight is better than artificial light. It is employed either in such a way that the patient sits, with his eyes closed, facing the sun, and the light is allowed to fall directly into the throat or so that the sunlight is thrown by the reflector into the throat. If the sunlight is glaring, we employ a special mirror with a longer focus (or a plane mirror), because the ordinary reflector would make a too glaring light, and sometimes even produce an uncomfortable sense of heat in the throat. Electric light is an excellent substitute for sunlight.

In making the examination we sit directly in front of the patient, have him open his mouth, set the reflector at the proper angle, then warm the laryngeal mirror a little over a spirit-lamp (testing its temperature by placing it against the back of the hand), have the patient put out the tongue, seize it with pieces of gauze, or a napkin or handkerchief and draw it out [as far as possible. It is well to have the head

[1] Let it be distinctly understood that what is here given contains only the most essential points which are of use in the examination itself. They cannot and should not take the place of study of these subjects in a medical course.

thrown quite well back]. Holding the mirror as one would a pen, it is to be slowly and carefully introduced into the mouth, and then the patient required to distinctly pronounce "æ," at the same time giving the proper direction to the mirror as it is pushed as far back as possible into the pharyngeal cavity, slightly pressing up the soft palate. The parts are now brought into view by elevating the mirror, depressing it, turning it now to the right, then to the left, and revolving it, both during quiet respiration and phonation.

The mirror must be most scrupulously cleaned and disinfected after every examination. It is not necessary to employ a special mirror with patients who are manifestly syphilitic.

Irritability of the pharynx (strangling, vomiting) may, with practice, be avoided. In very obstinate cases we can employ cocain. (See the special text-books regarding other obstacles and the ways of meeting them.)

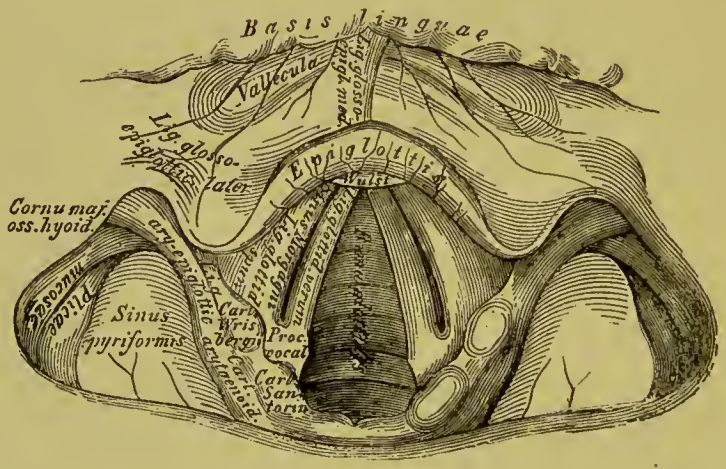


FIG. 182.—Laryngoscopic view during quiet breathing (after Heitzmann), double size.

In the laryngoscopic image the parts that are anterior appear as the posterior; on the other hand, what is upon the right hand of the patient remains upon the right; the examiner has, of course, the right vocal cord of the patient upon his left side.

We observe (see Fig. 182): 1. The base of the tongue, the glosso-epiglottic ligaments, the epiglottis, lig. aryepiglottica with the cartilages of Wrisberg. 2. The arytenoid cartilage, or the cartilage Santorini, the false vocal cords, the sinus Morgagni. 3. The ligamenta glottidis vera, with the vocal process of the arytenoid cartilage. 4. The region between the arytenoid cartilages, pars interarytenoidea (the posterior wall of the larynx); the subchordal region, or the foreshortened trachea. The illumination must be strong.

It is advisable for those who have had but little experience to first fix the landmarks by the shining white prominent true vocal cords, and from thence to examine the individual parts of the laryngeal picture one after the other.

The examination with the laryngeal mirror is directed to three things: the form and the color of the parts of the larynx, and the position or motion of those that move.

As to the form of the several portions of the inside of the larynx it is to be remembered that the representation given in Fig. 182 is, of course, only schematic. Repeated examinations of normal larynges will show the variations and fix them in mind. The form of the epiglottis varies very much; this is also true of the arytenoid cartilage and the false vocal cords or the opening of the ventricle of the larynx.

The color of the mucous membrane of the larynx, with the exception of the true vocal cords, is tolerably uniform and corresponds somewhat with that of the hard palate. Very often the upper border of the epiglottis, and sometimes its upper surface, is lighter, even yellowish-red. Above the arytenoid or Santorinian cartilages, the color of the mucous membrane varies considerably: sometimes it is exactly like the other parts, sometimes darker, again lighter, and then yellowish. The true vocal cords are shining white; in individual cases, with the function perfectly normal, they are slightly rosy. At the vocal process there is a circumscribed yellowish spot.

We must be on guard against being misled by deposits of mucus or of pus from the lungs. These deposits may be superficial, or may conceal deep ulcerations, loss of substance, croupous deposits. If in doubt, require the patient to cough. If still uncertain, have the patient inhale the vapor of steam for a few minutes, and then repeat the examination.

Normally the positions and movements of the portions of the larynx are perfectly symmetrical, although it is to be remarked that if the mirror is not properly held in position, the parts may easily appear to be unsymmetrical. During quiet respiration, the rima glottidis is tolerably widely opened—at least so that the whole breadth of the true vocal cords is visible; the arytenoid cartilage (cartilage of Santorini) can be seen between the pars interarytenoidea (posterior wall of the larynx); with active deep inspiration, the vocal cords separate from each other still more, so that they almost or quite disappear under the false vocal cords (which likewise stand apart). During phonation, the vocal cords come so closely together that either no slit between them, or scarcely any, can be seen. Generally their median edges form a perfectly straight line. But in individual cases, only the pares ligamentosæ close so sharply, and posterior to the process. vocales (that is, the pars cartilaginea), the vocal cords remain somewhat more apart, leaving a triangular space between them.

When the glottis is closed the arytenoid cartilages come near together and the pars interarytenoidea disappears; on the other hand, the false vocal cords leave a tolerably broad space between each other, through which we see the true vocal cords.

Pathological Conditions.—Since we here come upon a subject that has already been frequently referred to, in what follows we bring forward only those conditions which have relations to other internal diseases, and treat of them in the briefest way.

We do not meet with *paleness of the mucous membrane of the larynx* as a local condition. Also, it is no longer of importance in the recognition of a general anemia, because this is much easier determined by the paleness of the skin, lips, etc. Only one circumstance needs mention, that tuberculous infiltration and ulceration, in contrast

with other kinds, as syphilitic, often accompanies a very striking general paleness of the mucous membrane of the larynx. *Abnormal redness of the mucous membrane of the larynx*, without other changes, occurs in febrile hyperemia of all the mucous membranes and in general or local engorgement (the latter caused by pressure upon the larynx by tumors, from engorgement in the region of the cava superior). Also, whenever there is any redness of the larynx it must, as a matter of course, lead us to examine most carefully for any possible other changes (ulcerations, swellings, etc.).

Redness, swelling, and sometimes *secretion*, are the signs of *catarrh*. Acute as well as chronic laryngeal catarrh may involve various locations: for example, it may attack the upper portion of the larynx, leaving the glottis free; it may also attack only the glottis. A simple catarrh is always symmetrical. Acute, as well as chronic, catarrh may cause motor disturbances: on the one side this may be due either to the swelling of the mucous membrane (especially of the incisura interarytænoidea, preventing the closure of the glottis), or, to paralysis of the tensor of the vocal cords or the adductors. Acute laryngitis, especially in children, may give rise to apparent stenosis by reason of the swelling.

It is to be especially remembered that chronic and recurrent acute catarrh, and likewise, no doubt, simple catarrh, are very frequent in all chronic diseases of the lungs and especially in tuberculosis. It is further important to remember that behind a chronic catarrh a tuberculous or syphilitic (or lupous) new formation may for some time be concealed. A swelling which is limited to or elects the interarytenoid region is always very highly suspicious of tuberculosis.

Laryngitis hypoglottica (von Ziemssen) is an especially severe form of acute, as well as chronic, catarrh. In this disease we see beneath the vocal cords sometimes merely a soft rosy border, which can only be seen during inspiration; sometimes a firm grayish-red, smooth or uneven lump (see Fig. 183). It is almost always present upon both sides. These subchordal swellings appear to vary a good deal as to their nature: sometimes they are simply due to edema; in other cases, to a simple catarrh; in still others, to submucous infiltration. Further, such a subchordal laryngitis may be or may become tuberculous in its nature; more rarely it is syphilitic. From the condition of the larynx alone it is extremely difficult to make the differential diagnosis of these specific diseases from simple catarrh, as well as between syphilis and tuberculosis. There may, however, be other alterations of the larynx present, or unquestionable signs in other organs, which throw light upon the matter. The serious character of laryngitis hypoglottica is manifested by the fact that very frequently, and sometimes very suddenly, it causes severe stenosis.



FIG. 183.—Swelling below the vocal cords from laryngitis hypoglottica chronica (after Ziemssen).

Marked *swelling of the whole larynx* or of certain portions of it indicates edema or phlegmon—that is, severe submucous inflammation which ends in abscess. Both of these will chiefly be distinguished by

the color of the mucous membrane, which is pale when there is non-inflammatory edema, even yellowish and often shaking like jelly, while in phlegmonous inflammation it is deep red. Midway between these two conditions stands inflammatory edema, which pathologico-anatomically and genetically cannot be sharply distinguished from phlegmonous infiltration. Severe phlegmon may lead to decided disfigurement of the larynx (see Fig. 184). This may also be true of edema, as is shown in Fig. 185. Circumscribed laryngitis phlegmonosa usually results in the formation of abscess, or it may occasion a submucous or perichondrial formation of pus.



FIG. 184.—Phlegmonous laryngitis, with phthisical ulcer (from v. Ziemssen after Türck).

a, epiglottis; *b*, left aryepiglottic fold; *c*, left pyriform sinus.

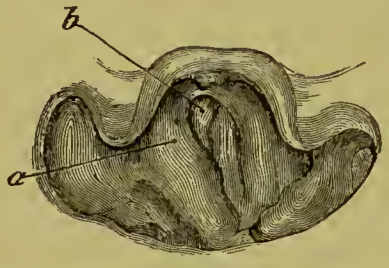


FIG. 185.—Extensive phthisical ulceration of the larynx, marked stenosis of the larynx from edema (from v. Ziemssen after Türck).

a, right aryepiglottic fold; *b*, anterior portion of the right cord.

Both these conditions are extremely dangerous, because they very easily result in stenosis, and sometimes, if they are acute, with remarkable suddenness. Phlegmonous laryngitis sometimes results in the formation of pus in the larynx (especially perichondritis), or its neighborhood (as angina Ludovici). Laryngeal cartarrh very seldom terminates as a phlegmon; foreign bodies, and substances that irritate chemically and as escharotics, may produce it; and lastly, it occurs in various acute infectious diseases, either resulting in catarrhal or ulcerative processes, or, it would seem, as independent metastatic diseases. Inflammatory edema may be the result in all of these cases, besides or instead of phlegmon. Simple edema is rare and chiefly occurs with general dropsy of all kinds, and in local obstruction (as in struma, mediastinal tumors).

Ulceration seldom occurs in the larynx with simple catarrh, more frequently in acute infectious diseases, especially in typhus abdominalis [typhoid fever] and variola, but most frequently in syphilis and tuberculosis. We limit ourselves to a description of the two last-named forms.

Syphilitic ulceration in the larynx occurs almost exclusively in connection with pharyngeal syphilis. It, by preference, attacks the upper section of the larynx, but it may appear in the glottis. In the majority of cases a single ulcer is observed. The ulcers have reddened edges, with a more or less shallow, whitish deposit upon a vocal cord or the epiglottis, or there is a very deep crater-like cavity with a whitish deposit and sharp or swollen border. By the absence of knotty elevations of the border they are sharply distinguished from carcinomatous ulcers. On the other hand, it is often difficult to distinguish

them from tubercular ulcerations. Here the differentiation is made by other signs of syphilis or tuberculosis that may be present.

Regarding *gummata* of the larynx, see below. Syphilitic infiltration without ulceration and without other associated signs of syphilis are very difficult to diagnose. These slighter syphilitic changes, moreover, very seldom come under examination, because they do not usually cause any inconvenience.[1]

Tubercular ulceration develops from tubercular infiltration. The principal location to be mentioned is the region of the interarytenoid space. The regions next most frequently attacked are the arytenoid cartilages and the false vocal cords. Tuberculous ulcers, with the exception of those upon the glottis, are more frequently multiple than are syphilitic. They are either very superficial and yellowish in color, or deep with swollen edges, sometimes, especially in the interarytenoid space, with papillomatous mucous proliferations. Although not pathognomonic (Gottstein), the latter form is in the highest degree characteristic of tuberculosis. Further, a pale edematous condition of the rest of the mucous membrane points to tuberculosis. The most important factor is the discovery of tubercle bacilli in the sputum. These may come from the larynx or from the lungs, which latter are always, or almost always, the first to be attacked.

Deep ulcerations may lead to perichondritis laryngea. The most frequent form is perichondritis arytænoidea. Perichondritis causes a very marked swelling and redness, generally over quite a large area. It very easily passes from this condition of swelling or collateral edema into stenosis. If it ruptures into the larynx, then the necrotic pieces of cartilage will be coughed out, and sometimes, when examining with the laryngeal mirror, we see them lying detached.

Scars are found in the larynx, as elsewhere, after healing from loss of substance. Those that chiefly interest us are the syphilitic. These, more than others, are inclined to retract, and hence they not infre-

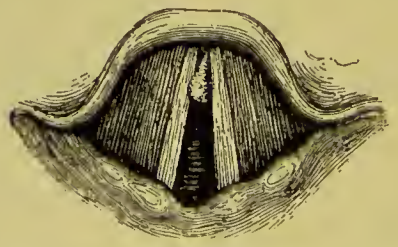


FIG. 186.—Pedunculated fibroma upon the under surface of the left vocal cord; position during inspiration (v. Ziemssen).

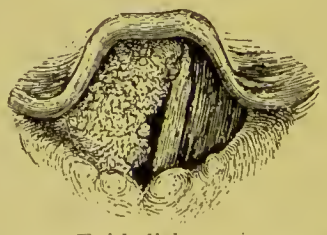


FIG. 187.—Epithelial carcinoma of the right vocal cord (v. Ziemssen).

quently result in stenosis. We either find a partial adhesion of the vocal cords or extensive cicatricial adhesions of the true and false vocal cords, with a funnel-shaped narrowing downward, etc. It is generally impossible to form any conclusion from the scar as to the nature of the antecedent processes. Only this, further, is to be said, that most laryngologists now agree that tubercular ulcers may cicatrize.

Excepting the syphilitic gummata, *new formations* in the larynx have only a local significance. Gummata are either solitary nodules

[1] See special works regarding them.

or a group of individually small nodules, at first red in color, with a crinkled contour. They are inclined to break up rapidly, and then to be replaced by deep ulcers.

The other new formations may be divided into benign and malignant. Of the former, very much the most frequent are the papilloma; more rare are the fibromata. Both, but especially the latter, are generally located upon the vocal cords. Papillomata are sometimes flat, wart-like, sometimes regular papules, often multiple, cauliflower-like. The fibromata are generally pedunculated; the surface is usually smooth, while that of the papillomata is uneven or villous. All the other benign new formations (lipomata, cysts, etc.) are extremely rare.

The malignant new formations are, in the great majority of cases, carcinomata. They, like the papillomata, generally develop from the vocal cords; next in frequency, from the false vocal cords. They manifests great inclination to necrosis and ulceration. The differential diagnosis of carcinoma, so long as there is no ulceration, is to be made from papilloma, after the occurrence of ulceration from tuberculosis and syphilitic ulceration: generally this is not easy. For particulars, we must refer to special works. Sarcoma of the larynx is much more rare than carcinoma.

In reference to the more unusual diseases of the larynx, like lupus and lepra, we refer to special works.

Spasm of the muscles of the larynx is not at all, or only exceptionally, observed with the laryngoscope. We here only mention phonic and inspiratory functional spasm of the glottis in adults. The former takes place at the instant when the effort at phonation is made, when a decided closure of the glottis takes place, as can be recognized with the laryngoscope; on the contrary, in the latter the vocal cords close at the instant of inspiration, hence, at the time when they ought to separate. During expiration the glottis is normal, or almost normally open, in opposition to paralysis of the [crico-arytænoidei] postici muscles,[1] in which they are very close together during expiration also.

As disturbances of coördination, both of these conditions will be understood from their analogy to the neuroses caused by certain occupations affecting the upper extremity (writer's cramp, etc.), and are to be accounted for by over-strain.

Paralysis of the Muscles of the Larynx.—*Paralysis of all the Muscles that Close the Larynx* (the crico-arytænoideus lateralis, arytænoideus transversalis, thyreo-arytænoideus ext. et internus—all supplied by the recurrent nerve). During phonation the vocal cords do not come close together, but remain in the position of inspiration. Complete aphonia is thus produced. The paralysis is generally bilateral, and is almost always due to hysteria as a basis. Hence, it is often combined with anesthesia of the larynx.

Paralysis of the Arytænoideus Transversus.—During phonation the most posterior portion of the glottis (the pars cartilaginea) does not close. As a result we have hoarseness, even to complete aphonia. It not infrequently occurs with acute laryngitis. (See Fig. 188.)

Paralysis of the thyreo-arytænoideus intern., one or both sides, causes

[1] See below.

imperfect closure of the glottis; when both sides are paralyzed there is a very narrow, symmetrical oval fissure (see Fig. 189); with unilateral

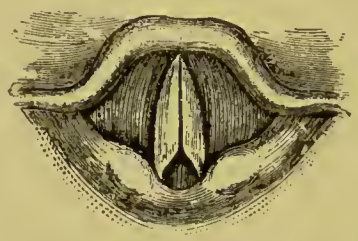


FIG. 188.—Paralysis of the arytænoideus in acute laryngitis (v. Ziemssen). The posterior portion of the glottis remains open during phonation.



FIG. 189.—Paralysis of both thyreo-arytænoidei interni, resulting from acute laryngitis (v. Ziemssen). Position during phonation.

paralysis, a correspondingly narrow, unsymmetrical fissure. It occurs in laryngitis, but, also, often in hysteria.

Paralysis of the crico-arytænoidei postici muscles, the openers of the glottis (recurrent nerve); posticus paralysis. The vocal cords in bilateral paralysis, during expiration, stand near together, and during inspiration still closer, sometimes in apposition; phonation may be quite normal. Hence, there is inspiratory dyspnea, with inspiratory stridor. The dyspnea may increase until there is asphyxia. In unilateral posticus paralysis the paralyzed vocal cord is motionless and lies near the middle line, while upon the sound side there are normal motions.

In its etiology, posticus paralysis is in many cases obscure. Sometimes it forms the beginning of a bilateral recurrent paralysis; in other cases it seems to have a muscular origin (gumma in the muscle, laryngitis with atrophy, etc.).

Recurrent paralysis—that is, paralysis of all the muscles supplied by the recurrent nerve—causes the vocal cords to assume the so-called cadaver position—the position with reference to each other that they have during quiet breathing. In severe paralysis, the vocal cords are entirely stationary in this position. In incomplete paralysis, they still make slight motions outward and also show an inclination to assume

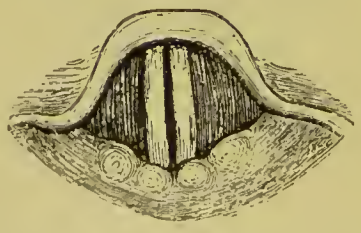


FIG. 190.—Bilateral complete posticus paralysis (paralysis of the crico-arytænoidei postici, dilatation of glottis) at the moment of inspiration (v. Ziemssen).

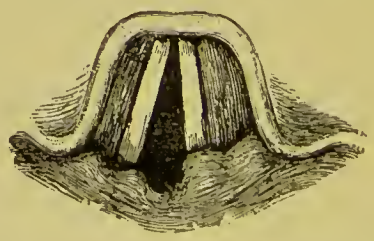


FIG. 191.—Position during inspiration in paralysis of the left vocal cord, or recurrent conduction paralysis (v. Ziemssen). Position and immobility of the left vocal cord, as in the cadaver.

the position of adduction, for which there is, as yet, no undisputed explanation. When the paralysis has continued for a long time the vocal cords become atrophied.

Bilateral recurrent paralysis produces bilateral cadaver position of the vocal cords, and thus complete aphonia and inability to cough. This is caused by compression of both recurrent nerves from aneurysm of the aorta, carcinoma of the esophagus, and enlarged glands. It will be readily understood that this bilateral paralysis from peripheral causes is much more rare than unilateral. Complete or incomplete bilateral paralysis of the recurrent nerve has been observed with bulbar paralysis, tumors, softening of the medulla, and compression of the vagi after their exit from the medulla.

Unilateral recurrent paralysis is much more frequent. It may be easily overlooked, because the voice is often clear, although weak, for the reason that the sound vocal cord during phonation reaches beyond the middle line. The paralyzed vocal cord during quiet breathing assumes the cadaver position, the sound one the position of rest—that is, somewhat more widely abducted than the other. During phonation the necessary closure of the glottis takes place, because the healthy vocal cord overreaches; but then the glottis is necessarily askew. Unilateral paralysis of the recurrent nerve is almost always due to compression of the nerve in the neck or as it passes into the thorax; this will be brought about by the same causes as bilateral peripheral recurrent paralysis. Thus, recurrent paralysis may be an important corroborative symptom of aneurysm, of carcinoma of the esophagus, or of any other kind of mediastinal tumor. When there is a suspicion of one of these conditions, we may almost regard a recurrent paralysis as decisive; at any rate, the existence of a recurrent paralysis has often given the first suggestion that led to a discovery of an aneurysm or of carcinoma of the esophagus.

Paralysis of the tensor of the vocal cords (crico-thyreoidei muscles, superior laryngeal nerve) is very seldom observed, and then it is always combined with anesthesia of the mucous membrane and paralysis of the epiglottis. It is a tolerably dangerous condition, because of the accompanying difficulty of swallowing and the risk of deglutition-pneumonia. The glottis, as viewed by the laryngoscope, is not exactly even, but wavy. In unilateral paralysis the normal vocal cord is somewhat higher than the paralyzed one.

Paralysis of the tensor of the vocal cords takes place most frequently in diphtheria, but then it is always accompanied with paralysis of other muscles.

2. RHINOSCOPY.

This is divided into anterior and posterior rhinoscopy. For anterior rhinoscopy we employ the reflector fastened to a band around the forehead, artificial light, a nasal speculum, and sometimes also a sound. Of the different nasal specula that of Jurasz is the simplest, but it requires both hands of the examiner, and hence occasionally it is necessary to use one of the complicated ones suggested by B. Fränkel, Duplay, Kramer, and others.

Anterior Rhinoscopy.—By this we see in the upright image the nasal septum, the lower and a part of the middle turbinated bone, together with the lower and middle nasal duct. The upper nasal duct and the upper turbinated bone are not visible. The nasal septum ex-

hibits a radish-yellow, more or less vertical, wall, with a more or less smooth surface; the turbinated bones are roundish convexities of a reddish color. In many cases we employ the sound in order to complete a diagnosis during illumination of the speculum. By the sound we ascertain the resistance, mobility or immobility of swellings, projections, tumors, foreign bodies, and move aside projecting polypi, etc.

The examination must often be preceded by a vigorous blowing of the nose, but only exceptionally is it necessary to use the nasal douche. Sometimes it is also necessary before using the sound to anesthetize the surface with cocain. This is accomplished by means of a very fine nasal sound armed with a pledget of cotton the size of a pea. This is dipped into a 10 per cent. solution of cocain (of which, however, the total quantity employed must not exceed a few drops), and then the liquid is applied with gentle pressure on the different parts of the mucous membrane. The cocain not only acts as an anesthetic, but it also reduces the swelling.

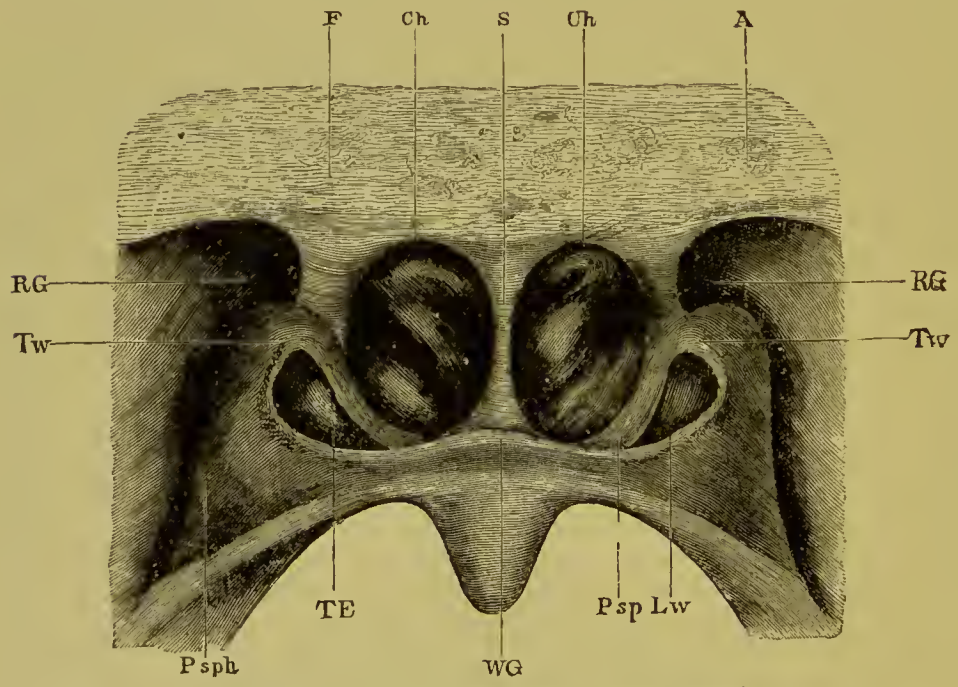


FIG. 192.—The rhinoscopic picture (after Schech).

S, septum narium, posterior nares with turbinated bones and nasal ducts: *WG*, posterior surface of the soft palate; *TE* and *Tw*, entrance into the tubes, eminences at the entrance; *Psp*, *Psph*, plica salpingopalatina and salpingopharyngea; *Lw*, levator swelling; *RG*, Rosenmüller's fossæ.

Posterior Rhinoscopy (Pharyngoscopy).—This is accomplished by a pharyngeal mirror illuminated by a reflector fastened on the forehead [as in the former proceeding]. During the examination the tongue must be held down with a spatula. The warmed laryngeal mirror is not held in the middle line, as in laryngoscopy, but alternately in the vault of the right and left palatal arches. It is not easy to interpret correctly the image of the naso-pharyngeal cavity, with the turbinated bones and nasal ducts, the Eustachian entrance, Rosenmüller's fossæ, and the roof of the pharynx. The difficulty is increased by the fact that frequently we cannot get an image of the whole at one time, and that the form of the respective parts differs in

different individuals. For the rest, compare the accompanying illustration [Fig. 192], taken from the excellent work of Schech.

In posterior rhinoscopy it may sometimes be necessary to palpate with the sound, and occasionally to palpate directly with the finger.

What is to be noted in practising rhinoscopy has in part been alluded to in what has already been said regarding the nose and throat. The most essential points are: deformities of the bony frame, alterations in the mucous membrane (acute and chronic inflammations, adenoid vegetations, specific exanthemata, or plaques [mucous patches], lupous alterations, etc.; ulcerations, enlargement of the erectile tissues of the nose, tumors of all sorts). Of course we cannot enter into details here.

3. OTOSCOPY.

For this there is required a hand mirror, perforated in the middle (after v. Tröltsch; also Türck's reflector, which for laryngoscopy lately is frequently supplied with means for putting in a handle so as to use it as a hand-mirror), and a set of metal ear-specula. The best source of light is diffuse daylight. If this cannot be had, any artificial light may be employed. Direct intense sunlight cannot be used.

Draw the ear backward and upward, and then insert the ear-speculum. Masses of secretion or of epidermis in the meatus externus, which interfere with the examination, may be carefully removed by means of an elongated pledget of cotton, or, if that does not suffice, by carefully washing it out with an ear-syringe and water or solution of borax at a temperature of 28° C. [82° F.]. Ear-pincers should only be used by practised hands. Other hindrances which stand in the way of an inspection of the ear-drum, or of the introduction of the ear-speculum, belong to pathology (foreign bodies, inflammation, or furuncles, ulcerations, new formations, exostoses, etc.).

If the obstacles mentioned are not present we may at once inspect the ear-drum by illuminating it with the mirror. It appears as a gray, or yellowish-gray, shining membrane, which upon closer examination reveals certain details.[1] On the anterior upper part is seen a white shining protuberance, the short process of the hammer, projecting more or less distinctly. Backward and downward from this runs a narrow band, which is the handle of the hammer; forward and downward from its lower end is to be seen a bright triangular figure: the triangular reflex of light, which is produced by light falling on the ear-drum, as the ear is here drawn in. Above the short process of the hammer, bounded by the anterior and posterior folds of the ear-drum, is the membrana Shrapnelli, a spot of special significance in many diseases of the ear-drum and of the middle ear.

If the ear-drum is very much retracted or atrophied we occasionally see in the posterior superior quadrant the long leg of the anvil and the posterior leg of the stirrup.

The pathological alterations recognizable by examination with the ear-speculum are the following:

Anomalies of vaulting, and these are: *retraction of the ear-drum* (compare Fig. 194), recognized by the handle of the mallet appearing

[1] Compare Figs. 193 and 194.

to be drawn backward and inward and, by the latter circumstance, seeming to be shortened, a condition often caused by obstruction of the Eustachian tube by adenoid growths in the throat; also by scars, coalescence with the inner wall of the cavum tympani; *bulging outward*

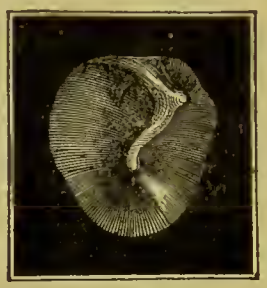


FIG. 193.—Normal right ear-drum (after Sarron).



FIG. 194.—Retracted left ear-drum (after Sarron).

of the ear-drum by mucous, serous, purulent exudations in the middle ear, by neoplasms, etc. in it.

Ruptures and perforations of the ear-drum: they may vary from an extremely minute size, scarcely to be recognized, to complete destruction of this membrane. Deposits of chalk, dulness, thickening of the ear-drum, are other changes.

Exudations in the cavum tympani: these are not always easy to recognize. Sometimes the level line of the exudation may be distinctly seen, and it changes with the change of position of the body, unless the effusion is encapsulated.

It remains to be mentioned that the diagnosis of coalescence of the ear-drum with the wall of the cavum tympani is best made by Siegel's pneumatic ear-speculum, or the simplified one suggested by Trautmann. It consists of a funnel, the exterior opening of which is closed by a glass plate. It can be put into the meatus auditorius air-tight. It is connected by a rubber tube to a rubber ball, by which the air in the funnel and meatus auditorius can be exhausted. Through the glass plate we may watch with the reflector the behavior of the ear-drum as the pressure in the meatus auditorius diminishes. When the coalescence is extensive it does not project forward at all, but with partial adhesion it projects somewhat.

As regards all special details we refer to special works upon diseases of the ear.

4. OPHTHALMOSCOPY.

This method of examination strictly belongs in the province of ophthalmology. Therefore we limit ourselves simply to its use for the purpose of diagnosis, where we observe a connection between certain changes of the fundus oculi and an internal disease.

(*a*) **Changes in the Fundus Oculi in Nervous Diseases.**—All diseases which lead to a general increase of the intracranial pressure may cause *choked disk* (*neuritis optica*). It is then always bilateral. At the same time choked disk may possibly be absent in all these conditions, but its presence is of the highest diagnostic significance, par-

ticularly in tumors and meningitis. Hydrocephalus is more rarely, and abscess of the brain very rarely, combined with choked disk. Unilateral choked disk is only caused by local pressure (a tumor, etc.) upon one optic nerve.

The extent to which vision is disturbed when we have choked disk varies very much; there may be none, or almost none. Disturbance of vision in choked disk usually occurs very early and markedly if the disease-process causes pressure upon the chiasm or the beginning of the optic nerve, as in tumors of the hypophysis cerebri, or, if there is hydrocephalus which presses inward upon the third ventricle (Wernicke). There must, of course, be disturbance of vision if the choked disk is followed by atrophy.

Pronounced choked disk is very easily recognized, yet it might be confounded with neuro-retinitis Brightii, which is exceptionally very much like it; but the exact recognition of a slight neuritis optica is very difficult. Whenever there is such a possibility an ophthalmologist should always be called in.

It seems that neuro-retinitis is particularly apt to be present in meningitis when there is a basilar exudation; it is also said to occur with encephalitis.

Primary atrophy of the optic nerve takes place (by the intraocular portion of the nerve changing into a white disk with a sharp boundary) especially in tabes, sometimes in multiple sclerosis, dementia paralytica; lastly, it occurs from pressure upon the chiasm. [The capillary circulation ceases, and hence the disappearance of the normal rosy hue.]

Finally, it is to be mentioned that *retinal apoplexy* has been observed as the forerunner of cerebral hemorrhage, emboli of the central retinal artery as the precursor of cerebral embolism. Regarding the changes of the fundus oculi in syphilis, see below.

We hardly ever find choroidal tubercle in tubercular meningitis. But it might occur with acute general miliary tuberculosis.[1]

(*b*) **Changes in the Fundus Oculi in Other Internal Diseases.**—*Retinitis* or *neuro-retinitis albuminurica*, with white specks, often arranged as radiating, sometimes confluent, lines around the macula, thickening of the walls of the vessels and hemorrhages, occurs particularly frequently in contracted kidney, also often in subchronic and chronic nephritis, but very seldom in acute nephritis. The disturbance of vision is greater or less according as the macula is attacked or not. Uremic amaurosis has nothing to do with this condition, but as a matter of fact this form of retinitis often occurs in uremia (and this is important for the diagnosis of this condition).

In *constitutional syphilis* (hence also in syphilis of the brain) we sometimes observe syphilitic changes in the fundus oculi: retinitis syphilitica, retinitis pigmentosa, choroiditis syphilitica.

We not infrequently find *tubercle of the choroid* in acute general tuberculosis, especially in the region of the macula; the tubercular deposits are generally very difficult to see.

In diabetes there occurs a peculiar so-called *diabetic neuro-retinitis* and *atrophy;* in leukemia, *hemorrhages* and *whitish exudate;* in per-

[1] See below.

nicious anemia, but also in simple, severe anemia, *hemorrhages* (generally easily seen).

Further, *retinal hemorrhages* are not unimportant diagnostic signs of pyemia, particularly pyemic endocarditis. They are not an absolutely fatal sign, as I myself saw in one case of puerperal pyemia: this undoubted case of pyemia, where besides the hemorrhages there were chills and slight icterus, recovered and the effused blood disappeared, leaving clear specks behind.

We have still to mention:

Pulsation of the retinal arteries in aortic insufficiency, *embolus of the central artery* in endocarditis (also frequently observed in chorea); lastly, after severe hemorrhages (particularly of the stomach, also of the intestine, and uterus) there occurs sudden *amaurosis*, not infrequently at first without any ophthalmoscopic conditions, afterward usually with distinct atrophy of the optic nerve.

Alcoholic amblyopia shows a negative condition, or else hyperemia, neuritis, atrophy; *tobacco amblyopia* shows a normal fundus oculi, or atrophy; in *amblyopia* or *amaurosis saturnina* either there is nothing, or else there is hyperemia and neuritic atrophy.

5. BACTERIA WHICH COME UNDER CONSIDERATION IN THE DIAGNOSIS OF INTERNAL DISEASES.

The object of the following sections is to summarize the notable peculiarities of the different micro-organisms which have already been mentioned in different parts of the work. But this pertains not only to the characteristics of the different organisms and their appearances when stained, but also to the much more important phenomena of their growth in cultures and under animal experimentation. In regard to the methods of procedure we must refer to the text-books upon bacteriology, and particularly to the instruction in the bacteriological courses.

Staphylococcus Pyogenes.—This consists of small round cells which are usually found in irregular masses, but are never arranged in chains. The spores of this, as of all other micrococci, have not yet been discovered. It grows upon gelatin even without much air, in the temperature of the room, still more rapidly and luxuriantly in a higher temperature. The gelatin becomes liquefied. Scratch cultures are either gold-yellow (Staph. pyogen. aureus), or white (Staph. pyogen. albus), or clear yellow (cereus), or citron yellow (citreus). Upon a surface it grows in round, light-brownish colonies looking like dots, which lose their sharp contour in the center of the fluid. Mice, guinea-pigs, and rabbits die in from two to nine days after intravenous and peritoneal injections. Mice are killed with certainty only after the subcutaneous injection of a large amount, but none of the other animals named are killed by subcutaneous inoculation.

It can be stained by all of the anilin stains, also by Gram's method. *It is the most common excitor of suppuration.* It is found in abscesses, furuncles, in many cases of empyema, purulent peritonitis; also in ulcerative endocarditis, etc., upon the valves of the heart; in pyemia and acute osteo-myelitis, in the suppuration which complicates typhoid fever, also exceptionally in erysipelas, etc.

Streptococcus Pyogenes.—This resembles the first named by its round cells, which form chains by progressive portions pushing out in the same direction, which sometimes twist around each other. The separate cells often vary in size. It grows slowly upon gelatin, better upon agar, in the temperature of the room, but more rapidly in an incubator at a temperature of 27° C. [= 80.6° F.]. It does not render gelatin fluid. The cultures upon a plate are extremely small, ½ mm. diameter, yellowish to yellowish-brown in color. When inoculated by puncture it develops slowly and does not spread out upon the surface of gelatin. It is stained like the preceding. It is fatal to animals only when they have been previously weakened; it causes redness and swelling of the rabbit's ear. There is frequently found a pus coccus which particularly inhabits the lymph tracts, and causes progressive phlegmon; it is also found in pyemia, especially puerperal pyemia, likewise frequently in endocarditis.

Streptococcus Erysipelatos.—Morphologically and as regards its staining qualities, it is like the preceding, but from the culture has thus far not with certainty been distinguished from it. In the rabbit's ear it causes a somewhat less active and extensive inflammation than the streptococcus pyogenes. The inflammation has the symptoms of erysipelas. Recently the opinion has become more and more prevalent that it is identical with the streptococcus pyogenes.

Micrococcus gonorrhœæ (gonococcus, compare Fig. 145, page 385). This usually occurs in the form of diplococci (roll-form), which often appear as tetracocci in that the single coccus has a bright stripe, as the beginning of a new portion. Culture of the gonococcus is somewhat difficult; until a short time ago it did not succeed at all. Bumm has cultivated the gonococcus upon coagulated blood serum in a moist chamber at a temperature of 32° C. [= 90° F.], and Gebhardt has bred it upon human blood serum; but the safest method is that suggested by Wertheim, who employs placental blood serum (or cyst fluid, ascites fluid) and meat-peptone-agar. Upon these and also upon agar to which has been added sterilized albuminous urine they grow vigorously from the pus of male gonorrhea; but in gonorrhea of the female culture is very difficult. However, culture is not requisite for diagnosis; we must even say that it by no means offers any greater security than the simple microscopic examination, because with culture mistakes may happen, and because the vaccination experiment has hitherto failed with animals, but upon men it could only exceptionally be permitted, and then only for scientific purposes.

Staining.—It is stained with all the anilin dyes, but best with Löffler's potassium-methylene-blue with heat; but the degree with which it takes the stain varies very remarkably. It is completely unstained by the Gram method. Cocci of the form and size of the gonococcus and contained within pus-corpuscles may be positively declared to be gonococci. If contained in epithelial cells they do not prove anything at all.[1]

Bacillus anthracis (see Fig. 90, page 246) is a rod, on an average about 5–10μ long, 1–1.25μ wide, with an abrupt end, often somewhat concave, with the inclination to develop into threads, without peculiar

[1] Compare Fig. 145, p. 385.

motion. It develops upon gelatin, potatoes, in alkaline urine at the ordinary temperature of the room, better at 36° C. [= 96.8° F.]. Sometimes there develop spores within spores (endogenous formation of spores). Gelatin is rendered fluid; when the amount of air is limited it develops poorly. Plate cultures, after twenty-four hours, can be seen, when slightly magnified, as round grayish-black spots, or wavy, as if curled; upon potatoes the cultures are gray-white, somewhat elevated. It is fatal to susceptible nursing animals (mice, rabbits, guinea-pigs, certain kinds of sheep), even with the most minute inoculation and in a very short time. They are found in capillary blood and in all organs richly supplied with blood, particularly the spleen; with living bodies they do not develop spores, likewise usually no long threads. They are stained by all basic anilin coloring-matters, but they are easily spoiled or become unclear if the covering-glass is made too hot; they become non-transparent if too strongly stained. They can also be stained by Gram's method.

Bacilli of malignant edema are 3–3.5μ long, 1–1.1μ wide (Flügge), hence thinner and shorter than the anthrax bacilli, from which they are also distinguished by the rounded ends. They form rigid threads, often of considerable length. The individual bacillus forms spores, and these are so large that they distend the bacillus. In the dependent drops they manifest peculiar motions. They only grow when oxygen is excluded, hence are anerobia. They develop in a reagent-glass, best in gelatin to which is added a one or two per cent. solution of grape-sugar (Flügge). They flourish best at the temperature of the body. But they only grow at the lower end of a deep, very fine canal formed by sticking in a needle, and this canal is to be again closed. It fluidizes the gelatin and forms an offensive-smelling gas. It is stained by all the anilin dyes, but poorly after the Gram method. It is found in garden soil, in muddy water, in the blood of asphyxiated animals, etc. A little of the soil taken up on the point of a penknife and put under the skin of the abdomen of a guinea-pig or rabbit generally kills it in one to two days by the invasion of the bacilli (but sometimes, during this experiment, tetanus develops). In man it causes edema and sometimes emphysema of the skin (see page 50).

Typhus abdominalis bacilli (see Fig. 134, page 351) [*bacilli of typhoid fever*] are short, slender rods with rounded ends, thrice as long as broad, one-third as long as the diameter of a red blood-corpuscle. They have active motions (hanging drops). They form threads in cultures and hanging drops, but not in living animal bodies. It is questionable whether they form spores. They develop, at the temperature of the room, upon gelatin, agar, without the character of the growth being characteristic. The culture upon potatoes or upon potato-gelatin shows important peculiarities (Holz): for some days after the inoculation it would seem as if nothing had grown—at most that the surface of the potato around the inoculating scratches has a moist shimmer; in the whole circumference of this shimmer a very thick resisting turf of bacilli is present. This characteristic inconspicuousness for the unassisted eye of a well developed culture is not always present. And this bacillus cannot yet be positively distinguished from the bacillus coli notwithstanding the most varied morphological and biological, also

biochemical characteristic criteria have been employed. The typhoid bacillus is best stained with carbol-fuchsin or Löffler's alkaline methylene-blue solution ;[1] it is to be washed only with water. It is discolored after Gram's method. It regularly occurs in the intestine, spleen, liver, kidneys, also in the stools, from which, however, it cannot always be freed, in the urine, and now and then in the blood in abdominal typhus [typhoid fever].

Widal's Reaction.—The typhoid bacillus has the extremely important peculiarity that, bred in bouillon culture, after the addition of a minute quantity of blood-serum from a patient ill with typhoid fever, or convalescent from it, within a few seconds it loses its peculiar movements and collects into heaps, lines, and gradually also into lumps which, if examined in hanging drops, are visible to the naked eye. The blood-serum of healthy persons, or of persons suffering from any other disease, does not have this effect upon the typhoid culture.

It seems that this highly interesting reaction has an extraordinary significance for the diagnosis of present or past typhoid fever. The reaction can be made with a few drops of blood taken from the skin.[2]

Colon Bacilli (Bacterium coli commune).[3]—This is a constant inhabitant of the intestinal canal from the first hours of extra-uterine life. It is a bacterium distinguished by varying morphological and biological behavior, and particularly by varying virulence. It occurs in intestinal catarrhs often in great quantities, even in pure cultures like the cholera bacillus, and is then very virulent for animals. It also occurs in the most varied parts of the body, particularly in the abdominal organs (peritoneum, gall-bladder, bladder, and renal basin), and there excites different degrees of inflammation. It shows a great similarity to the typhoid bacillus, with which many think it is identical. It is stained like that bacillus, and is easily bred upon gelatin.

Diphtheria Bacilli[4] (Löffler).—They are plump, partly curved small rods, distinguished by peculiar granules and curious club-shaped swellings at their ends. They do not thrive below 22° C. [71.6° F.], and they perish at 60° C. [140° F.]. The culture material hitherto used is generally the following mixture: 3 parts of blood-serum, 1 part of bouillon to which is added 1 per cent. of peptone, 1 per cent. of grape-sugar, and 0.5 per cent. of common salt. Deyke, however, has suggested a preferable alkali-albuminate-agar, which contains 1 per cent. of alkali-albuminate, 1 per cent. of peptone, 0.5 per cent. of common salt, 1½ to 2 per cent. of agar, and 5 per cent. of glycerin, and is exactly neutralized with pure HCl solution, then alkalized with 1 per cent. of a 33 per cent. solution of soda, the latter being added after the mixture has been filtered through cotton. It is best to place small pieces of membrane upon recently coagulated plates of Deyke's-agar.[5] Diphtheria bacilli morphologically resemble a great many other bacilli

[1] 30 c.cm. of concentrated alcoholic solution of methylene-blue, 100 c.cm. of 0.1 per cent. solution of potassium hydrate.

[2] See also p. 247. [3] Compare p. 352. [4] Compare Fig. 96, p. 261.

[5] Note in correction of the text: It is necessary to state that B. Fränkel, in a publication which has just appeared (Deutsche Vierteljahrsschr. f. öff. Gensundh.—Pflege, 1897). "Uber d. Bekämpfung der Diphtherie" recognizes as alone adapted to the purpose Löffler's sugar-serum and Tochtermann's serum-agar. We have had excellent results in over 300 cases with Deyke-agar.

(pseudo-bacilli of diphtheria—Hoffmann, xerose-bacilli—Ernst and others), but they are often safely to be recognized in the diphtheria membrane by their great quantities and peculiar arrangement.[1] These occur in the pseudo-membranes of diphtheria patients (constantly), and in their oldest parts, which are abundantly infiltrated with cells, never in internal organs. They have been found in the mouth in children after recovery from diphtheria and also exceptionally in healthy people. They are absent in scarlet-fever-diphtheria, in which, on the contrary, a chain coccus is found, if there is no mixed infection with bacillary diphtheria. It is pathogenic for rabbits, guinea-pigs, pigeons, and chickens; when introduced into the trachea they cause pseudo-membrane; in guinea-pigs if introduced under the abdominal skin other characteristic phenomena and generally death in four days. They are well stained with Löffler's methylene-blue and after Gram's method.

Influenza Bacillus (R. Pfeiffer).—This is a very small bacillus, difficult to stain, which appears in the sputum of influenza patients in great quantities, arranged in nests and often lying together by twos. They seem to be the exciters of influenza, since they regularly appear in this disease, and often as almost the only micro-organism in the sputum. They are stained with difficulty. Cover-glass preparations are laid for five to ten minutes upon a pale-red watery dilution of carbol-fuchsin. Culture is difficult upon nutrient material containing hemoglobin.

Tubercle bacilli (compare Fig. 52, page 159, and Fig. 144, page 384) are thin rods, 1.5–3.5μ long (Flügge), frequently slightly curved or somewhat broken; they often form threads, and sometimes two or more lie closely together. Very often they contain a number of egg-shaped spaces (spores?), and then, when stained and slightly magnified, they sometimes look like chain micrococci. They have no independent motion. They grow best in a reagent-glass upon an oblique coagulated, sterilized blood-serum and glycerin-agar, at a temperature of 37.5° C. [100° F.] (min. 30, max. 42). At best they grow very slowly, and hence precautionary measures are necessary that it may not develop excessively.[2] In fourteen days there appear small, dull-white scales and specks, which, when slightly magnified, show an arrangement that reminds one of a tangled braid of hair.[3] We can have it develop upon a covering-glass and then stain it by the method described on page 248. [See also Methods of Staining described on page 160 *ff.*] The experiment of inoculation is best made upon guinea-pigs, by placing some sputum, for instance, in the abdominal cavity. Generally, there is no reaction in the peritoneum. After two or three weeks the glands swell, and in four to eight weeks the animal dies.

Lepra bacilli resemble small tubercle bacilli, and are stained with anilin in the usual way, but also like tubercle bacilli; hence, like the former, there may be a double staining. They are found in leprous skin, in the glands, in the tissue-juices, in the nerves, also said to be found in the blood, etc.; they occur mostly in small and large cells resembling giant-cells. Cultures have not yet been successfully made.

Anthrax bacilli are like tubercle bacilli, only somewhat broader.

[1] See Fränkel's publication referred to above. [2] For the technique, see special works.
[3] Compare Fig. 144, p. 384.

They are stained with Löffler's potassic methylene-blue. Stain very carefully, then wash with dilute acetic acid. They are often easier and more certainly demonstrated by culture than by animal experimentation. They develop rapidly upon slices of potato at 35° C. [= 95° F.], as a brownish, slimy mass. It can be inoculated upon guinea-pigs; some maintain that puppies are better. Death follows after an indefinite time, and nodules occur in various organs (one of the first symptoms is a swelling of the testicles).

The **cholera bacillus**[1] has been very fully described upon page 350*f*. It has there been pointed out that the certain proof is only made by culture. A mucous floccule from the stools or from the linen is taken, and is used for plate culture, either at once, or after enrichment of the possible cholera vibriones. The latter procedure is rather to be recommended.

By "enrichment" is understood the production of a luxurious growth of cholera bacilli on the surface of a fluid nourishing solution. This facilitates and accelerates the diagnosis of cholera. The flock of mucus is placed in a Dunham's solution of peptone. This solution consists of a sterilized solution of 1 per cent. peptone, 0.5 per cent. of common salt, of a strongly alkaline reaction. After six hours, kept at a temperature of 37° C. [98.6° F.] there is a pronounced turbidness of the upper stratum of the liquid which is produced by a pure culture of the cholera bacillus ("membrane formation"). In most cases practised observers will be tolerably certain in regard to the matter; but absolute certainty is only attained by making plate cultures from this membrane. Agar plates at 37° C. require 8 to 10 hours; gelatin plates (5 to 10 per cent. culture gelatin) which can be kept only at 22° C. [71.6° F.] require one to two days; small white points are seen in the depth, which gradually come to the surface, and by liquefaction of the gelatin in the depth produce deep funnel-shaped depressions. At the bottom of the funnel there are white cultures no larger than the head of a pin. The infecting puncture made into gelatin in a test-tube undergoes a change as follows: a funnel appears here also, which in its upper part contains a large air-bubble formed by the rapid evaporation of the fluidized gelatin. The lower portion of the puncture resembles a thin thread which, in places, looks clear like glass, like an empty capillary tube, while at other places the cumulated, sunken culture appears as gray or whitish threads. In the hanging drop very lively movements, like a swarm of gnats, are seen. The bacilli show a predilection for the edge. Larger plate cultures, if slightly magnified, show a peculiar luster and arrangement as if they were composed of broken glass. How to transmit them: 1. Intra-peritoneal infection of guinea-pigs, according to R. Koch, will give a perfectly characteristic picture of the disease. 2. The inoculation is made upon guinea-pigs whose stomach-contents are made alkaline by 5 c.cm. of a 5 per cent. solution of soda (using an esophageal catheter); the intestine is made quiet by injecting into the peritoneum 1 c.cm. tinct. opium for each 200 grams weight [of the animal]; then, by means of the esophageal catheter, there is introduced 10 c.cm. of the deposit of the cholera bacilli in bouillon. After two days the animal dies (often without

[1] See Figs. 130-132, pp. 350, 351.

diarrhea, always without vomiting): the condition of the intestines is found to be exactly like that in cholera. In the intestines are abundant cholera bacilli.

The Cholera Reaction (Nitrozo-indol-reaction).—Recently this has again come into notice as a means of diagnosing cholera, but it does not seem to have an independent diagnostic value.

Bacilli of Finkler-Prior (see Fig. 133, page 351) resemble cholera bacilli, but are thicker and plumper; but in the colored preparations they cannot certainly be distinguished from Koch's comma bacillus. Plate cultures develop remarkably rapidly, and render gelatin fluid in much larger quantity than cholera bacilli. This difference in the rapidity of development is the best mark of distinction. When slightly magnified, the cultures seem to be very finely and uniformly granular, of a yellowish-brown color. The inoculation-puncture, likewise, shows a much more rapid fluidization, but not the clear threads beneath the upper "air-bubble," but an irregularly wide channel, which reminds one of a stocking. After a week the whole test-tube becomes fluid. Also, the inoculation of animals give a different result—stinking intestinal contents, while in cholera they smell stale.

Bacillus Enteritidis.—This was found by Gärtner in Frankenhausen in cases of meat-poisoning, and later by others (Karlinski). It is probably a frequent exciter of meat-poisoning; possibly it may be the only exciter. It is a short, thick little rod, surrounded by an areole, which grows best upon nutrient gelatin. The cultures have a light-gray, coarsely granulated, transparent appearance, and do not liquefy. It has been found in the spleen of a person who died from poisoning, and in the meat which had caused the poisoning (Gärtner), in the stool and vomit of another case (Karlinski), but it has also been found in the intestinal contents of healthy persons. Raw meat had a more poisonous effect, but cooked meat also in part caused sickness. If introduced subcutaneously, the bacillus caused death to mice, rabbits, guinea-pigs.

The bacillus enteritidis is stained by all the anilin dyes. In staining the coloring matter lies in the middle; the ends of the bacillus are free from stain.

INDEX.

ABDOMEN, diminished volume of, 278
distention of, circumscribed, 278, 282
in disease of peritoneum, 281
drawing in of, 278
scaphoid, 279
fluctuation of, 283
increase of dimensions of, 278
inspection of, in diseases of peritoneum, 281
measuring circumference of, 283
puncture of, to draw off ascitic fluid, 285
scaphoid, 279
sound on percussing, 97
topography of, 268
Abdominal breathing, 74
disappears in paralysis of diaphragm, 80
contents, position of, illustrated, 269
disease, headache with, 447
retardation of pulse in, 204
distention, effect on manner of breathing, 282
inflammation restricts action of diaphragm, 80
muscles, paralysis of, 495
pressure, function of, 496
reflexes, 458
reflex, increase of, in intercostal neuralgia, 458
unilateral absence of, 533
swellings, sensation of resistance over, 102
typhus. See *Typhoid fever.*
wall in emphysema, 75
Abelmann on effect of extirpation of pancreas on fat-digestion, 348
on resorption of food in amyloid degeneration, 335
Abnormal constituents of urine, 393
sound over lungs, 109
Abscess, deep, shown by edema, 48
due to dentition, 254
irregular enlargement of liver from, 289
of abdominal wall, pain from, 278
simulating meteorism, 278
of anus, pain at stools in, 335
of brain, choked disk in, 547
of chest-wall, weakened vocal fremitus with, 134
of liver, fluctuation of, 292
surface of organ in, 292
tenderness in, 290
of lungs, effect of, on thorax, 78
elastic fibers in sputum of, 152
hematoidin in sputum in, 147
lung-tissue in sputum in, 147
Abscess of lungs, purulent sputum in, 145
of spleen, enlargement of organ in, 298
tenderness in, 298
of tonsil, 259
peptonuria with, 398
perinephritic, detection of, 357
retropharyngeal, cyanosis in, 38
detection of, 260
subphrenic, 284
Abscesses, peptonuria in, 398
staphylococcus pyogenes in, 548
Absolute deadness, 96
galvanometer, 465
Absolutely deadened sound, 96
Absorption by stomach, 307
Abulia, 439
Accidental blood-murmurs, 197 (note)
Accommodation in breathing, 85
of valvular deficiency, 169
paralysis of, 515
Aceto-acetic acid in urine, 373
Acetone in urine, 409
odor of, 373
with diaceturia, 407
test for, 409
Acetonuria, 409
Achorion Schönleinii in vomit, 332
Achroödextrin from starch, 304
test for, 321, 322
Acid-curve of urine, 364
-intoxication in diabetic coma, 408
Acidity of urine, determination of, 373
Acids, salivation caused by, 256
Acoustic amnesia, 506
nerve, affections of, disturbance of hearing in, 523
Acromegalia, 531
Actinomyces, cause of suppurations in mouth, 257
in pleural exudation, 138, 139
in sputum, 150, 164
Active spasm of muscles, 456
Acuoxylon, value of, 120
Acute exanthematous diseases, 44
Addison's disease, bronze skin in, 43
Adenoid growths in throat, drawing-in of ear-drum in, 546
Afanassiew on secretion of bile-pigment in poisoning, 41
After-sensibility, 443
Ageusis, 524
Agrammatismus, 513
Agraphia, 506
literal, 513

Agraphia, varieties of, 513
with ataxic aphasia, 507
Air in peritoneal cavity, auscultation and percussion of, 285
in peritoneum, causes diminished area of liver-dulness, 296
-passages, stenosis of, diminished inspiratory pressure in, 141
swallowing of, 310
Akataphasia, 513
"Albumin curve," 396
determination of, in pleural exudations, 139
in cerebrospinal fluid, 435
in serous sputum, 145
in urine, 378, 393
in health, 366
qualitative tests for, 394–396
rare forms of, 398
quantitative test for, 396
Albuminometer, 397
Albuminous corpuscles in sputum, 164
Albuminuria, 378
cyclic, 394, 396
genuine, when occurs, 394
inosite with, 406
peptone with, 398
renal, casts in urine in, 380
when occurs, 394
retinitis with, 547
transitory, 394
Albumoses in urine, 398
Albumosuria, 398
Alcohol, absorption of, by stomach, 357
-amblyopia, central scotoma in, 521
immoderate use of, effect on disease, 21
-paralysis, diminished irritability in, 485
-poisoning, glycosuria in, 401
headache in, 447
odor from mouth in, 253
use of, effect on disease, 21
Alcoholic amblyopia, appearance of eye in, 548
stools, color of, 337
odor of, 336
tremor, 489
Alcoholism, trembling of tongue in, 254
Aldehoff on casts in urine of diabetic coma, 380
Alderton, recoil-theory of apex-beat, 172
Alexia, 506
Algosis faucium leptothricia, 263
Alkalies, salivation produced by, 256
Alkaline fermentation, odor of stools of, 336
of urine from micro-organisms, 383
reaction of urine in health, 364
Allochiria, 443
Amaurosis after severe hemorrhages, 548
saturnina, ophthalmoscopic appearance of eye in, 548
temporary partial, significance of, 522
uremic, 547
Amblyopia, alcoholic, appearance of eye in, 548
saturnina, appearance of fundus in, 548
tobacco-, appearance of fundus oculi in, 548
Ammonia in urine in diabetes, 408
-poisoning, odor of vomit in, 332
Ammoniacal fermentation of urine, 363
stool, 336
Ammoniaco-magnesian phosphate, crystals of, in feces of typhoid fever, 349
in urine, 389
Amnesia, acoustic, 506
phenomena in, 508
testing of, 509
visual, 506
Amnestic aphasia, 507, 508
Amphoric breathing, 126
Amphoteric reaction of urine, 365
Amyl-nitrite poisoning, glycosuria in, 401
Amyloid degeneration of gastric mucosa, anacidity in, 325
of intestine, poor resorption of fats in, 335
kidney, albuminuria in, 394
hyaline casts in urine of, 380
liver, increased consistence of, 292
surface of, 291
spleen, enlargement of, 298
Amylolysis, disturbed, in superacidity of stomach, 325
incomplete, shown by microscopical examination of vomit, 331
Amylolytic period of digestion, 305
Amyotrophic lateral sclerosis, 454
bladder disturbance absent in, 528
increased tendon reflexes in, 461
reaction of degeneration in, 484
Anacidity of stomach, 309
significance of, 325
Analgesia, 443
Anamnesis defined, 17
form for recording, 26
method of obtaining, 19
what it comprises, 18, 20
Anarthria, 502
Anasarca, 46
Anatomical diseases, symptoms of, 533
Anchylostoma duodenale, 344
Anchylostomiasis, 345
Charcot's crystals in feces of, 349
Anemia a cause of dropsy, 47
albuminuria in, 394
alteration of percentage of hemoglobin in, 238
ascending venous pulse in, 229
caused by bothriocephalus latus, 343
color of urine in, 369
dicrotic pulse in, 213
diminution of alkalescence of blood in, 250
of red corpuscles in, 238
dizziness in, 471
frequent pulse in, 207
gravis, blood-corpuscles in, 241
headache in, 447
heart-murmurs in, 197
hemorrhages in fundus of eye in, 548
leukocytosis in, 242
local redness in, 38
microcythemia in, 240
oligocythemia in, 238
pernicious, alteration in red corpuscles in, 240
leucin and tyrosin in urine of, 390

Anemia, retinal hemorrhage in, 514
uric acid increased in, 392
poikilocytosis in, 241
polyuria in, 366
produced by anchylostoma duodenale, 345
redness of face in, 37
signification of the term, 231
small pulse in, 209
sounds over arteries in, 222
splenic, enlargement of spleen in, 298
strengthening of heart-sounds in, 187
subjective sensibility of hearing in, 523
sensations of vision in, 522
syncope in, 438
venous humming in, 230
vertigo in, 438
Anemic heart-murmurs, 97
necrosis of pons and medulla, 422
Anesthesia, 443
cause of, 420
of mucous membrane in hysteria, 458
of pharynx, 527
sensory, in gross hysteria, 491
unilateral, 443
Aneurysm, aortic, pulsation in heart-region in, 176
blood in sputum from rupture of, 144
contraindicates use of stomach-sound, 313
difficulty in distinguishing from apparent enlargement of heart, 181
increase in size of chest in, 141
neuralgia due to, 447
of abdominal aorta, 219
of aorta a cause of cyanosis, 38
and other arteries, effect of, on pulse, 211
crystals in sputum in hemorrhage into lungs from, 156
cyanosis from, 38
distinguished from empyema pulsans, 90, 91
phenomena in, 218, 219
pressure by, upon recurrent nerve, 543
pulse-curve in, 216
of arch of aorta affects vessels of left side, 219
of ascending aorta affects vessels of right side, 219
of descending aorta, 219
of innominate artery, 219
of minute arteries of brain, hemorrhage from, 526
of pulmonary artery, 219
pressing heart forward, 176
pressure on nerves from, 436
pulsation of, near stomach, 272
simulates enlarged heart, 181
small pulse in, 209
Angina, coronary, bradycardia in, 204
enlargement of tongue in, 254
herpetica, 45
lacunar, tonsils in, 258
Ludovici a cause of cyanosis, 38
pus in larynx from, 539
Ludwigii, detection of, 255
pectoris, bradycardia in, 204
frequent pulse in, 207
tonsils in, 258
Angle, epigastric, 73
of Louis, 73
in emphysematous thorax, 75
in phthisical thorax, 76
Angulus Ludovici, 68, 73
Anidrosis, 33
Anilin-poisoning, color of blood in, 233
Animal parasites in sputum, 157
in urine, 382
of alimentary canal, 340
Ankylostomiasis. See *Anchylostomiasis.*
Ankylostomo-anemia, edema in, 48
Anodal closure, result of, 467, 468
Anomalies of respiration, 79
Anorexia, 439
Anosmia, 524
Anterior gray columns, disease of, reaction of degeneration with, 484
horn of cord, characteristic sign of paralysis above, 456
excitability of, increases skin-reflex, 458
results of disease of, 454
tendon reflexes decreased in disease of, 461
rhinoscopy, 543
Anthrax bacilli, 552
in blood, 246
Anticipating intermittent fever, 63
Antifebrin in urine, test for, 411
Antimony in urine, 411
Antiperistalsis of stomach, 272
Antipyrin in urine, test for, 411
Anuria, 367
in hysteria, 528
nervous, 368
Anus, examination of, 280
fissure of, pain at stools with, 335
Anxiety, cause of disease, 21
frequent pulse in, 207
increases secretion of urine, 362
mydriasis in, 517
Aorta, anatomical relations of, 218
aneurysm of, phenomena in, 218, 219
pulse-tracing in, 216
pulsation of, 218
sclerosis of. See *Sclerosis.*
stenosis of. See *Stenosis.*
phenomena in, 219
various phenomena of, 218
Aortic aneurysm, pulsation in heart-region in, 176
heart-sound, second, strengthening of, 188
insufficiency. See *Insufficiency.*
stenosis. See *Stenosis.*
effect of, 168
Ape-hand, 499
Apex-beat, alteration of width and strength of, 173
cause of, 172
disappearance of, 174
displacement of, 172
doubling of, 175
examination of, 171
strength and breadth of, a sign of hypertrophy of left ventricle, 174
weakening of, 174

Apex-curve, 212
Aphasia, 506
amnestic, 507, 508
atactic, 507
causes of, 507
phenomena of, 507
testing for, 509
ataxic, 506, 507
Charcot's diagram of, 510
graphic representation of, 509–513
literal, 507
motor, 507
sensory, 506
Aphasic disturbances, 503
mode of testing, 509
Aphemia, 507
Aphonia, 67
from recurrent paralysis, 543
how caused, 541
tone of cough in, 143
Aphthæ in vomit, 332
Aplasia of lung, deadened sound over, 100
Apnea in Cheyne-Stokes' respiration, 81
Apoplectic habit predisposes to hemorrhage of brain, 525
Apoplexia cerebri, glycosuria from, 402
Apoplexy, albuminuria with, 394
coma from, 437
decubitus after, 530
from tinnitus aurium, 523
glycosuria with, 402
imbecility from, 438
pulse in, 210
Apparatus for electrical examination, 462
Appendicitis, pain on palpation of right iliac fossa in, 277
Apple-odor of urine, 373
Arbutin, color of urine after taking, 371
Arc de cercle, 434, 491
Arhythm, 207
Arm, motor-centre for, 416
motor-points of, 470, 471
muscles of, paralysis of, 498
Aronsohn-Phillips's stain for neutrophile cells, 245
Arsenic in urine, 411
-paralysis, diminished irritability in, 485
-poisoning, jaundice from, 42
Arsenical melanosis, 43
Arseniuretted hydrogen, poisoning by, causes hematohepatogenous icterus, 401
Arterial liver-pulse, 228
pressure, increased, polyuria in, 366
sclerosis. See *Sclerosis.*
Arteries, auscultation of, 221
examination of, 201
normal condition of, 221
palpation of, 220
pathological conditions of, auscultation of, 222
sclerosis of, a cause of hypertrophy of left ventricle, 170
Arthritis deformans, pain in spine in, 447
stiffness of spinal column in, 434
Fränkel's pneumococcus in, 163
Arthropathiæ in nervous diseases, 530
Arytænoideus transversus, paralysis of, 541
Ascarides in ductus choledochus a cause of jaundice, 41
Ascaris lumbricoides, 343
eggs of, 345
Ascites, 281
absence of liver-dulness in, 296
appearance of skin in, 282
associated with cirrhosis of liver, 291
chylous, 286
diagnosis of, 281
effect on form of chest, 77
enlargement of veins of abdomen in, 282
expansion of thorax in, 77
from venous engorgement, 223
that moves about, 282
Asiatic cholera. See *Cholera.*
vomit in, 328
Aspergillus fumigatus in sputum, 149
in sputum, 164
Aspermatism, 412
Asphyxia, local, in nervous diseases, 526
Aspirated fluid, examination of, 139
Aspiration emphysema, 49
Associated movements, 492
Asthma, bronchial, a cause of cyanosis, 38
cause of emphysema of lung, 89
Charcot-Leyden crystals in, 149, 157
Curschmann's spirals in, 155
expiratory dyspnea in, 88
frequent respiration in, 84
spiral casts in, 149
mucous threads in sputum of, 150
oxalate of lime in sputum of, 157
simulation of, 20
spirals in sputum of, 150
uremic, 399
Atactic aphasia, 507
causes of, 507
phenomena of, 507
testing for, 509
Ataxia, 487
disturbance of conception of motion in, 446
handwriting in, 513
how shown, 487
of upper extremity, 513
when it occurs, 488
Ataxic aphasia, 506, 507
Atelectatic crepitation, 131
Atheroma of aorta, systolic murmurs with, 218
of arteries, pulse-curve in, 214
of blood-vessels, causes disease of vessels of brain, 526
Athetosis, 492
Atonic atrophic paralysis, 457
degenerative paralysis, 419
paralysis, 457
Atony of stomach, symptoms in, 324
Atrophic paralysis, 453
fibrillary contractions in, 490
Atrophy, acute yellow of liver, diminished urea in, 392
diminution of phosphates of urine in, 392
disappearance of liver, dulness in, 296
leucin and tyrosin in urine of, 390
and paralysis, parallelism between, 454, 455
circumscribed, 453

Atrophy, degenerative, 453
diffuse, 453
puerile muscular, increased motility of spinal column in, 434
muscular, from joint-disease, diminished irritability in, 485
myopathic muscular, diminished irritability in, 484
of inactivity, 453
of muscles, 453
of optic nerve, concentric narrowing of field of vision in, 521
in tabes dorsalis, 513
mydriasis with, 517
primary, 547
of paralyzed muscles as a symptom, 533
of tongue, 494
primary myopathic, 455
progressive muscular, 454
fibrillary contractions in, 490
increased irritability in, 485
spinal progressive muscular, decreased tendon reflexes in, 461
reaction of degeneration in, 479, 484
Atropin, effect of, on pupil, 517
-poisoning, red skin from, 37
Attitude of patient, 28
Auditory tract, 421
Auenbrugger, the discoverer of percussion, 92
Aura of epilepsy, 490
Auriculotemporal nerve, distribution of, 448, 449
Ausculation, direct, 119
indirect, 119
mediate, 119
methods of, 119
of arteries, 221
of heart, 182
points of election for, 184
of intestine, 281
of lungs, 118
history, 118
of peritoneal cavity, 285
of pulse, 221
of stomach, 276
of veins, 230
of voice, 133
when it may be omitted, 135
of whispered voice, 135
stethoscopic, 119
Autocthonous thrombi, effect on pulse, 211
Auto-intoxication with aceto-acetic acid, 407
with acetone, 409
by oxybutyric acid, 407, 408
Auxiliary muscles of respiration, 496
Axillary artery, compression of, fulness of veins of arm in, 224
lines, 69
nerves, illustrated, 450
Azoöspermia, 412
temporary, 413

Baas and Penzoldt's explanation of puerile breathing, 122
Baccelli's auscultation of the whispered voice, 135
Bacilli in blood, 245
Bacilli in cerebro-spinal fluid, 435
Bacillus anthracis, 549
comma, in vomit of Asiatic cholera, 351
enteritidis, 352, 554
of anthrax, 552
of Asiatic cholera in feces, 350
of cholera, 553
of diphtheria, 551
of Finkler-Prior, 554
of glanders in blood, 247
of influenza, 552
of lepra, 552
of malignant edema, 550
of tuberculosis, 552
in blood, 246
in feces, 353
in pleural exudations, 138
in sputum, 150, 159
of typhoid fever, 351
ureæ in urine, 383
Back, percussion of, 104
Bacteria, casts of, in urine, 382
diagnostic value of, 548
in feces, 349
in mouth-contents, 257
in pleural exudations, examination for, 137
in urine, 361
in vomit, 332
intestinal, in stools, 349
of digestion, 305, 306
Bacteriological examination of blood, 247
Bacterium coli commune, 352, 551
in urine, 383
Finkler-Prior in feces, 352
Balz on distoma pulmonum in sputum, 149, 158
Bamber, reference to, 166
Band-box note, 116
Barrel-shaped crystals of uric acid in urine, 387
Bartels on wandering kidney, 357
Basch (V.), sphygmomanometer, 210
Base of brain, effect of lesions of, 427
Basedow's disease, blowing murmur over thyroid glands in, 223
frequent pulse in, 207
hemidrosis in, 34
palpitation and pain of heart in, 526
sensations of heat in, 526
strengthening of heart-sounds in, 187
strengthened heart-beat in, 174
tremor in, 489
Basilar artery, 421
Basophile cells, staining of, 244
Baths, cold, value of pulse in showing effect of, 218
Baumann on relation between cystin and ptomaïns in urine, 390
Beale on carbonate of lime in urine, 389
Benczúr and Jonás, determination of demarcation of organs by surface temperature, 91
Benedict's symptom-complex, 427
Berger, microbe of whooping-cough, 164
β-oxybutric acid in urine, 407
Biceps-muscle, electrical stimulation of, 471
Biceps-tendon reflex, 460

Biedert's method of demonstrating tubercle bacilli in sputum, 161
Bienstock, reference to, 349
Biermer, reference to, as improver of percussion, 92
on "band-box note" in emphysema of lungs, 116
Biermer's change of sound, 117
Biernacki on decrease of hydrochloric acid in nephritis, 324
Bile-acids in urine, 400
in health, 366
coloring-matter in urine, 371
deficiency of, absorption of fat in, 336
diminished flow of, stools in, 337
in stomach-contents, 315
-pigment in sputum, 147
in urine, 400
vomiting of, 328
Biliary colic, vomiting in, 326
engorgement, tenderness of liver in, 290
Bilious stools, 337
vomit, 328
Biot's respiration, 83
Bismuth test for sugar in urine, 402
Bitter almonds, poisoning by, odor of vomit in, 332
Black-water fever, formations in blood of, 249
Bladder, atony of, residual urine in, 368
crises in tabes, 528
distended, diagnosis of, 360
disturbance of, points to palpable disease, 533
examination of, 360
importance of emptying, in examining abdominal organs, 303
paralysis of, 528
position of, 360
tenesmus of, 361
"Bleating voice," 135
Blood, abnormal additions to, 245
alkalescence of, 250
amount of, in body, 231
anthrax bacilli in, 246
bacilli in, 245
casts in urine, 382
chemical examination of, 250
color of, 232
in poisoning by different substances, 233
coloring-matter of, in urine, 370
consistency of, determination of, 236
corpuscles, counting of, 238
in feces, 348
in urine, 375, 377
in vomit, 331
red, number of, in cubic millimeter of blood, 238
normal size of, 240
altcration of in malaria, 249
alterations in number and appearance of, 238
in size and form of, 240
in sputum, 151
staining of nuclei of, 241
white, counting of, 244
in saliva, 256
in sputum, 151
Blood-corpuscles, red, in stomach-contents, 316
in urine, 377, 378
proportion of to red corpuscles, 242
normal and pathological condition of, 241–244
determination of amount of hemoglobin in, 233
examination of, 231
filaria sanguinis hominis in, 250
glanders-bacillus in, 247
icterus, 41
in cerebrospinal fluid, 435
in stomach-contents, 315
in stools from hemorrhage of stomach, 329
in urine, appearance of, 375
makes it turbid, 371
microscopical examination of, 377
reaction of urine with, 373
in vomit, testing for, 330
malarial parasites in, 247
method of obtaining for examination, 232
microscopic examination of, 237
micro-organisms in, 245
-pigment in urine, tests for, 400
-preparation, dry, making of, 237
making of, 237
methods of drying, 244
staining of, 244
-pressure, reduced, anuria in, 268
quickness of coagulation of, 250
specific gravity of, determination of, 236
spectroscopic examination of, 235
spirillum recurrens in, 246
-supply of brain, 421
testing for, in vomit, 330
tubercle bacilli in, 246
typhoid bacillus in, 246
uric acid in, 250
vomiting of, 328, 329
watery condition of, polyuria in, 366
Bloody sputum, 145
stools, 338
vomit, 328, 329
Blue disease, 38
-red skin, 38
Blushing, 37
Boas on resorcin as a test for hydrochloric acid in stomach, 317
Body, normal temperature of, 53
temperature of, 50
Boilliaud, reference to, 506
Bone-conduction, testing of, 522
-reflexes, 460
Bones and joints, anomalies of, in nervous diseases, 530
arrest of growth of, after paralysis, 530
brittleness of, in tabes, 530
sound on percussion of, 96
Borborygmi, 281
Bornhardt on relation of weight of body to height, 32
Bothriocephalus latus, 342
Boulimia, 439
Boundaries of lungs, extension of, 117
changed condition of, 117

Bowels, disturbance of, from general venous engorgement, 224
involuntary discharge of, 335
non-tympanitic sound over, 99
obstruction of, grass-green vomit in, 328
tympanitic sound over, 98
Brachial artery, auscultation of, 221
plexus, paralysis of, 450
Brachialis anticus, electric stimulation of, 471
Brachycephalus, 431
Bradycardia, 203
occurrence of, 284
Brain-abscess, fever in, 57
base of, effect of lesions of, 427
blood-supply of, 421
-disease, Cheyne-Stokes' respiration in, 82
disturbance of consciousness in, 437
retardation of pulse in, 204
symptomatic convulsions in, 490
topographical diagnosis of, 423
organic diseases of, imbecility in, 438
Braune on physiological accentuation of angle of Louis, 73
Breath, odor of, 252
Breathing, abdominal, 74
disappearance of, from paralysis of diaphragm, 80
alteration of, in disease of larynx, 67
amphoric, 126
anomalies of, as regards pregnancy and rhythm, 81
of manner of, 80
asymmetry of, 80
audible, 87
bronchial, 121
costal, 74
diaphragmatic, 74
difficult, 29, 83
failure of action of diaphragm in, 80
metamorphosing, 127
normal, 73
obstructed, 67
of compression, 126
painful, effect of posture upon, 29
puerile, 122
thoracic, replaced by diaphragmatic, 80
transition, 127
undefined, 127
vesicular, 121
Brenner on electric examination of acoustic nerve, 522
Brick-dust sediment in urine, 363, 387
Brieger on connection between ptomaïns and cystin in urine, 390
Bright's disease, dropsy with, 399
edema in, 48
Brittleness of bones in tabes, 530
Broca's aphemia, 507
Bromin in urine, test for, 410
Bronchial asthma. See *Asthma.*
from nasal affections, 525
breathing, 121
as a symptom of diseases, 125
distinguishing of, from vesicular breathing, 123
catarrh. See *Catarrh.*
Bronchical tubes, closure of, weakened vocal fremitus in, 134
Bronchiectasis, aspergillus fumigatus in sputum of, 150
friction-sound in, 133
leptothrix granules in, 150
odor of sputum in, 147
quantity of expectoration in, 144
Bronchitis a cause of cyanosis, 38
acute croupous, tube-casts in, 149
mucous sputum in, 144
alveolar epithelium in sputum of, 151
capillary, non-uniform crepitation in, 132
change of vesicular breathing in, 124
Charcot-Leyden crystals in, 157
chronic, leptothrix granules in, 150
croupous, tube-casts in, 149
Curschmann's spirals in, 155
leptothrix buccalis in sputum of, 150
croupous, Charcot-Leyden crystals in, 149
cyanosis from, 43
dry râles in, 129
fetid, crystals of fatty acid in sputum of, 156
mucopurulent sputum in, 145
odor of sputum in, 147
tyrosin in sputum of, 157
ferment in sputum of, 164
frequent respiration in, 84
inspiratory drawing-in of, 87
jerking respiration in, 125
moist râles in, 130
prolonged expiration in, 124
purulent, coin-shaped sputa in, 145
vital capacity of lungs in, 141
Bronchophony, 135
Bronchoblennorrhea, quantity of expectoration in, 144
Bronchus, stenosis of, effect of, 87
Bronze skin, 43
Bruit de pot fêlé, 116
Bulbar dysarthria, 502
paralysis a cause of cyanosis, 39
absence of bone-reflexes in, 461
absence of pharyngeal reflex on, 458
cyanosis in, 39
diminished irritability in, 485
disturbance of speech in, 502
increased secretion of saliva in, 527
paralysis of diaphragm in, 80
paralysis of recurrent nerve with, 543
position of soft palate in, 494
quickened pulse in, 526
reaction of degeneration in, 484
speech in, 502
pulse, 227
Bulbus jugularis, engorgement of, 225
Bulging in neighborhood of heart, 175
Bumm, method of cultivating micrococcus gonorrhœæ, 549
Burns, hemoglobinuria after, 370
"Buzzing" felt near heart, 177

CACHECTIC leukocytosis, 242
Cachexia a cause of general atrophy, 454
cancerous, oxaluria in, 389
chronic, disturbed consciousness in, 437

Cachexia, defined, 31
delirium in, 438
diminished urea in, 392
fever in, 59
indicanuria in, 369
oxaluria in, 389
paleness in, 36
state of skin in, 32
tuberculous, oxaluria in, 389
Cachexias as a cause of atrophy, 454
Cadaver position of vocal cords, 542
Cadaveric odor of breath, 253
Calculi in urine, 391
phosphatic, 391
urinary, residual urine in, 368
testing of, 391
Calculus, mulberry, 391
renal, thickening and distention of ureters in, 360
Callus, pressure on nerves by, 436
Camman, stethoscope of, 120
Cancer. See *Carcinoma.*
-navel, upon liver, 292
of stomach, tumors of, 273
Cancrum oris, detection of, 255
Cane-sugar, digestive changes in, 304
Cantani on oxaluria, 389
Cantharides poisoning, fibrin in urine of, 398
Capillary pulse, 220
Capsule, inner, lesion of, with hemichorea, 492
Caput medusæ, 225
quadratum, 430
Carbolic acid, color of urine after taking, 371
in urine, 411
poisoning, odor of vomit in, 332
Carbonate of lime in urine, 389
Carbonic acid, absorption of, by stomach, 307
inflation of stomach with, 271
poisoning, glycosuria in, 401
Carbonic-oxid poisoning, spectroscopic appearance of blood in, 235
Carcinoma, acetone in urine in, 409
coma from, 437
nutrition poor in, 525
of esophagus, pressure by, upon recurrent nerve, 543
of gall-bladder, tumor in, 292
of kidney, palpation of, 358
shreds of tissue in urine of, 379
of larynx, 68, 541
of liver, density of organ in, 292
surface of liver in, 291
tenderness in, 290
of pancreas, palpation of, 302
of peritoneum, 283
of pleura, hemorrhagic exudation in, 139
of skull, 431
of spleen, surface of organ in, 298
enlargement of organ in, 298
of stomach, anacidity in, 325
hypacidity in, 324
hypersecretion in, 325
white blood-cells in stomach-contents in, 316
of tongue, hardness of tongue in, 254
ulcerating, odor of vomit in, 332
Carcinoma ventriculi, bloody vomit in, 329
peptonuria with, 398
villosum of bladder, shreds of tissue in urine of, 379
Carcinomatous coma, 437
pleurisy, cells in exudate of, 137
Cardia, stenosis of, detection of, 267
of stomach, position of, 268
Cardiography, 175
Caries of teeth, foul odor from, 252
of spine, stiffness of spinal column in, 434
pain in spine in, 447
sensitiveness of spine to pressure in, 434
Cartoid artery, auscultation of, 221
pulsation of, in health, 220
left, importance of relation of, to aorta, 423
pulse in aneurysm of aorta, 219
Cartilage in sputum, 148
sound on percussion of, 96
Case-taking, value of, 25
Caseous crumbs in urine, 379
Casts, epithelial 382
granular, 381
hyaline, 380
in urine, 379
the infallible signs of nephritis, 378
of bacteria in urine, 382
of hemoglobin in urine, 382
renal, kinds of, 380
preparation of, for examination, 380
waxy, 380
Catalepsy, 492
Cataleptic rigidity, 492
Cataract with diabetes mellitus, 514
Catarrh, atrophic gastric, anacidity in, 325
bronchial, diminished vesicular breathing in, 124
dry râles in, 128
chronic gastric, superacidity in, 325
relation to nervous disease, 525
intestinal, diarrhea in, 336
pain on pressure in, 277
nasal, as a symptom, 66
of apices of lungs, dry râles in, 129
of bronchial tubes, dyspnea in, 85
of intestine, bacterium coli commune in, 352
bilious stools in, 337
distention of abdomen in, 278
epithelial cells in stools of, 348
fat in feces in, 348
intestinal infarction in, 338
mucous stools in, 337
reaction of stools in, 336
watery stools in, 338
of larynx, appearance of larynx in, 538
of stomach, condition of stomach-contents in, 311
detection by test-meal, 314
hypacidity in, 324
hyperacidity in, 325
hypersecretion of, 325
microscopic appearance of stomach-contents in, 316
of trachea, cylindrical epithelial cells in sputum of, 151

Catarrh, ulceration of larynx in, 539
Catheter, use of, a cause of cystitis, 528
Cathodal closure, 467
opening, 467
Cat's purring, 196
Caudate nucleus, blood-supply of, 422
Causes of disease, 21
Celli, method of staining malarial parasites in blood, 248
Celsius's thermometer, 50
Centers, motor, 416
of special senses, 420
reflex, 419
Centigrade thermometer, 50
Central fiber of Curschmann's spiral, 154
motor tract, lesions of, effect of, 424
nervous system, injuries of, glycosuria in, 402
paralysis, arrest of growth of bones after, 530
scotoma, when occurs, 521
vomiting, 326
Centrifuge for examining urine, 374
Centripetal tracts, 420
Cephalalgia, 446
Cercomonas in sputum, 158
Cerebellar ataxia, 488
Cerebellum, disease of, vomiting in, 527
Cerebral abscesses generally multiple, 532
affections, ataxia in, 488
artery, middle, importance of, 422
"blowing," 221
congestion with uremia, 399
nerves, course of, 418
paralyses, increase of tendon reflexes in, 461
paralysis of children, hemiathetosis in, 492
peduncle, effect of lesions of, 427
tumors, Cheyne-Stokes' respiration in, 82
Cerebrospinal fluid, examination of, 435
meningitis. See *Meningitis.*
Cerebrum, diseases of, atrophy from, 456
Cervical spinal cord, injury of, fever in, 525
veins, diastolic collapse of, 229
vertebræ, caries of, a cause of defective motility of head, 495
Cestodes, 341
Chain-cocci in urine, 383
Charcot on pressure on hysterogenous zone arresting an hysterical spasm, 529
stigmates hysteriques, 533
Charcot's crystals in feces, 349
in leukemic blood, 244
diagram of aphasia, 510
Charcot-Leyden crystals, 149
in sputum, 156
Chest, alterations of form of, by deformity of skeleton, 78
asymmetry of, 73
delineation of cross-section of, 140
pathological forms of, 75
pigeon, 78
rachitic, 78
-wall, thickenings of, vocal fremitus in, 134
Cheyne-Stokes' respiration, 81
cause of, 83
dyspnea in, 84
in brain disease, 82
Cheyne-Stokes' respiration in uremia, 399
Chiasm, pressure on, primary atrophy of optic nerve in, 547
Chill described, 56
Chloral-poisoning, glycosuria in, 401
Chlorate-of-potash poisoning, color of blood in, 233
icterus from, 42
Chlorid-of-iron reaction for diacetic acid, 407
of urine, 373
Chlorid of sodium in urine, 365, 392
Chloroform-poisoning, icterus from, 42
odor from mouth in, 253
Chlorosis, ascending venous pulse in, 229
color of blood in, 233
of urine in, 369
diminution of percentage of hemoglobin in, 238
Egyptian. See *Egyptian Chlorosis.*
frequent pulse in, 207
heart-murmurs in, 197
sounds over arteries in, 222
strengthening of heart-sounds in, 187
syncope in, 438
venous humming in, 230
Choked disk, 513
as a symptom, 533
disturbance of vision by, 547
easily confounded with neuroretinitis, 547
significance of, 546
Cholemia from long-standing jaundice, 41
Cholera, Asiatic, anuria in, 367
bacillus of, 350
habitat of, 350
mode of preparing cultures, 350
diarrhea in, 334
indicanuria in, 369
mucous vomit in, 328
rice-water stools in, 338
watery stools in, 338
bacillus, 553
in feces, 350
increase in amount of feces in, 335
reaction, value of, 554
stools, odor of, 336
sweat in, 34
Cholesterin-crystals in sputum, 156
Chordal paralysis, unilateral ageusis in, 524
Chorea, disturbance of conception of motion in, 446
embolus of central artery of retina in, 548
minor, 441
Choroid, tubercle of, 547
Choroidal tuberculosis in acute miliary tuberculosis, 514
Choroiditis syphilitica, 547
Chrysophanic acid, discoloration of urine by, 371
Chylous ascites, 286
Chyluria, 371
caused by filaria sanguinis hominis, 382
fibrin in urine of, 398
lipemia in, 245
lipuria in, 406
Chyme, hydrochloric acid in, determination of, 320

Ciliated epithelium of trachea, transfer of mucus by, 143
Circle of Willis, 422
Circulation, disturbance of, casts in urine in, 380
Circulatory apparatus, examination of, 166–251
relation of disturbances of, to nervous disease, 526
Circumpolarization for determining sugar in urine, 406
Cirrhosis of liver, alcoholic, urobilinuria in, 369
ascites in, 282
bloody vomit in, 329
color of urine in, 369
disappearance of dulness in, 296
enlargement of spleen in, 298
increased consistence in, 292
surface of liver in, 291
tenderness in, 290
Citron on proportion of albumin in pleural exudations, 139
on Reuss's formula for estimating albumin, 140
Clavus hystericus, 447
Claw-hand, 499
Clear percussion-sound, 94
Clinical law of contraction, 467
Clod-voice, 502
Clonic spasms, 488
when occur, 490
Closed tympanic sound, 98
Cloudiness of urine in disease, 371
Coalescence of ear drum with wall of tympanum, 546
Coal-soot in sputum, 147
Coal-tar preparations, color of urine after taking, 371
Coating of tongue, 255
Cocain, effect of, on pupil, 517
Cod-liver oil, lipuria after taking, 407
Coffee-grounds stools, 339
vomit, 329
Coffin-lid crystals in urine, 389
Cohnheim on cause of edema, 48
on polyuria in diabetes, 367
reference to, 47
Cohnheim's hypothesis of dropsy in kidney-disease, 399
Cold bath. See *Bath*.
Colic, bradycardia in, 204
from gall-stones, 339
renal, 391
Collapse, cold sweat of, 34
described, 57
sudden fall of temperature in, 59
weakening of apex-beat in, 174
Colon bacilli, 551
distention of, for diagnostic purposes, 280
determination of boundary of, by inflation, 280
position of, illustrated, 269
relation of, to spleen, 299
tumors of, confounded with those of spleen and kidney, 279
Color-sense, testing of, 522
Columns of Clarke, 420
of Türck, 417
Coma, breathing in, 81
carcinomatous, 437
definition of, 437
diabetic, apple-odor of urine in, 373
casts in urine of, 380
caused by oxybutyric-acid poisoning, 407, 408
diaceturia with, 407
difficulties of diagnosis in, 437
in diabetes mellitus, 528
post-epileptic, 490
uremic, 399
Combined system-disease, 532
Comma bacilli in vomit of Asiatic cholera, 351
Compensation of valvular insufficiency, 169
Compensatory hypertrophy of heart, 169
Compression of spinal cord, herpes zoster in, 529
Compulsory positions, 30
Concealment of disease, 19
Concentrated urine, appearance of, 363
Conception of localization, testing of, 444
of space, testing of, 445
Concretions in urine, 391
Concussion of brain, glycosuria from, 402
Conductive resistance, determination of, 474
Conductivity of bones for sound, testing of, 522
Confluent sputa, 145
Congestion of liver, urobilinuria in, 369
Congo-red test for hydrochloric acid in stomach, 316
Conjugate deviation, 514
diagnostic signification of, 516
Conjunctiva, color of, 35 (note)
nerve supply of, 448
Consciousness, disturbances of, 437
Consolidation of lungs a cause of cyanosis, 39
Consonant râle, 130
Constipation, 333
alternating with diarrhea, 334
and severe obstruction to be sharply distinguished, 334
habitual, in nervous disease, 527
Constrained positions and motions, 491
Constricted liver from tight lacing, 290
Constriction, feeling of, about thorax, 446
Consumption, dry cough in, 143
Contents of stomach, examination of, 303
Continued fever in the course of febrile diseases, 61
range of temperature in, 56
Continuous secretion of gastric juice, 311
Contracted kidney, retinitis albuminurica, 547
Contraction, cathodal closure, 467
opening, 467
clinical law of, 467
electrical, normal laws of, 468
Contractions, fibrillary, 489
idiomuscular, 485
paradoxical, 486
produced by electric stimulation of nerves, 468

Contractures from paralyses, 457
of muscles in paralysis, 457
Conus arteriosus, sound caused by filling of, 184
terminalis, location of, 434
Convalescence indicated by weight of body, 31
perspiration in commencement of, 34
pulse in, 202
temporary imbecility in, 438
Convergent strabismus, 514
Convolutions of brain, illustrated, 415
Convulsions, epileptic, described, 490
glycosuria after, 402
opisthotonos in, 434
hysterical, 491
opisthotonos in, 434
infantile, from eruption of teeth, 254
with uremia, 399
Cooing in intestines, value of, 281
Co-ordinated spasms, 491
Co-ordination and ataxia, 486
how acquired, 486
of speech, test for, 418
temporary loss of, 488
Copaiva, odor of urine after taking, 373
Copper-poisoning, teeth and gums in, 254
Coracobrachialis, function of, 498
Cord, spinal, compression of, by kyphosis, 434
Corona radiata, 417
effect of lesion of, 425
Corpora quadrigemina, ataxia from lesions of, 468
Corpus striatum, effect of lesion of, 427
Cortex cerebri, lesion of, effect of, 424
motor centers of, 419
Cortical center, lesions of, effect of, 425
Corvisart, revived percussion, 92
Costal breathing, 74
Cough, 142
as a sign of disease, 142
character of, in disease of larynx, 67
dry, 143
frequency of, 142
in whooping-cough, 143
moist, 143
nervous, 525
reflex, 142
suppression of, 142
time of day when most apt to occur, 142
tone of, 143
vomiting from, 143
with tough expectoration, 143
Coupland on value of record-keeping, 25
Cracked-pot sound, 111, 116
moist, 116
Cranial nerves, location of nuclei of, 424
points of exit from skull, 424
Cranium, diminution of size of, 431
enlargement of, 430
examination of, 430
form of, 431
injuries and concussions of, disturbance of consciousness in, 437
sensibility of, to pressure, 433
size of, 430
Creasote, color of urine after taking, 371
Cremaster reflex, 458
Crepitant râles, 131
Crepitation, 131
atelectatic, 131
indux and redux, 131
non-uniform, 132
Cretinism, 438
Crico-arytænoidei postici, paralysis of, 542
Crico-thyreoidei, paralysis of, 543
Crises, gastric, 527
laryngeal, 525
Crisis defined, 56, 61
protracted, 61
Critical perturbation, 61
Crossed paralysis of eye, significance of, 517
reaction, examination of, 518
Croup a cause of cyanosis, 38
casts of trachea and larynx in, 149
dyspnea in, 84, 85
inspiratory drawing-in of, 87
laryngitis in, 68
tone of cough in, 143
Crural artery, auscultation of, 221
Crus cerebri, tumor of, effect of, 426
Crutch-paralysis, 451
Crystals in sputum, 149, 155
in stools, 349
of fatty acid in sputum, 156
of hematoidin in sputum, 155
Cubebs, odor of urine after taking, 373
Curare-poisoning, glycosuria in, 401
Current-changer, 466
-strength, irritating, 463
measurement of, 464, 465
total, 463
Currents, electric, measuring of, 464, 465
Curschmann's spirals, 154
origin of, 155
Curvature of spine, lateral, recognition of, 434
Curve at the base, 212
Cutaneous reflexes, 457
sensibility, 439
veins, enlargement of, 224
Cyanosis, 38
causes of, 38
color of tongue in, 254
from general venous engorgement, 224
from severe coughing, 143
in general neuroses, 526
in tetanus and epilepsy, 496
of new-born, 38
Cyclic albuminuria, 394, 396
Cylindroids, diagnosis of, from urinary casts, 375
in urine, 375
Cyrtometer, 140
Cystic hemorrhage, appearance of blood in urine from, 376
Cysticerci in brain and eye, 527
Cystin, concretions of, in urine, 391
in urine, 390
Cystitis, alkaline fermentation of urine in, 383
cylindroids in urine of, 375
from cystin in urine, 390
from ptomaïns in urine, 390

Cystitis from use of catheter, 528
hematuria in, 376
in nervous affections, 528
increased mucus in urine of, 375
micro-organisms in urine of, 383
odor of urine in, 373
pus in urine of, 377
reaction of urine in, 373
signs of, 361
sulphuretted hydrogen in urine in, 374
tenesmus of bladder in, 361
turbid urine in, 371
white blood-corpuscles and pus in urine of, 377
Cystopyelitis, thickening or tenderness of ureters in, 359
bacterium coli commune in, 383
Cystopyelonephritis, bacterium coli commune in, 383

DAHMEN'S method of demonstrating tubercle bacilli in sputum, 161
Damoiseau's curve, 111
Dax on speech-disturbance, 506
Deadened resonance, 109
sound, relatively, where it occurs, 100
when heard, 99
Deadness applied to sound, 94
absolute and relative, 95, 96
percussion, 94
Deafness, testing for, 522, 523
Death-agony, breathing in, 81
Decubitus, 530
acutus, 530
disturbance of nutrition with, 525
malignus, 530
ordinary, 530
Deep breathing, employment of, in examining abdomen, 290
percussion sound, 95
sensibility, testing of, 444
Defervescence of fever, 59
Deforming arthritis in hemiplegia and tabes, 531
in syringomyelia, 531
Degeneration of cord, character of, as determined by location of lesion, 425
of nerves and muscles, 480
reaction of, 478. See *Reaction of degeneration.*
Deglutition-pneumonia from paralysis of tensor of vocal cords, 543
Dehio on origin of puerile breathing, 122
on proportion of hemoglobin to number of red corpuscles in phthisical and carcinomatous cachexia, 239
table of variations of examinations of blood with Fleischl hemometer, 234
Delayed sensibility, 443
Delirium, 438
in gross hysteria, 491
muttering, 438
tremens described, 438
with uremia, 399
Deltoid muscle, electrical stimulation of, 471
paralysis of, effect of, 497
Dementia, hand-writing in, 513
Dementia paralytica, primary atrophy of optic nerve in, 547
senilis, 438
Dentition, disturbances due to, 254
epileptiform attacks during, 490
Dermatograph, value of, 103
Desaga's spectroscope, 235
Detritus in stools, 348
Deviation, conjugate, 491, 514
of the eye, 514, 515
primary, 515
secondary, 515
Dextrin, absorption of, by stomach, 307
Diabetes, absence of free hydrochloric acid from stomach in, 324
acetone in urine of, 409
odor from mouth in, 253
casts in urine in, 380
coma from, 437
decipiens, 367
diaceturia with, 407
diminished alkalescence of blood in, 250
salivation in, 256
dyspnea in, 525
gangrene in, 526
increase of urea in, 392
insipidus in nervous diseases, 528
inosite in urine of, 406
lipemia in, 245
local asphyxia in, 526
mal perforant in, 530
mellitus, apple-odor of urine in, 373
caries of teeth in, 253
cataract in, 514
color of urine in, 369
grape-sugar in urine, 401
high specific gravity of urine in, 372
levulose in urine of, 406
nervous diseases in, 528
oxalate of lime crystals in urine of, 388
neuralgia in, 447
neuroretinitis in, 547
oxalate of lime in sputum of, 157
oxybutyric acid in, 407
perforating disease of foot in, 530
polyuria in, 367
symmetrical gangrene in, 526
thirst in, 23
Diabetic coma, acetone odor from mouth in, 253
alteration of vesicular breathing in, 124
dyspnea in, 525
diaceturia with, 407
Diacetic acid in urine, 407
Diaceturia, 407
Diagnosis defined, 17
etiological, 17
individual, 18
Diagnostic value of symptoms in nervous disease, 531
Diaphanoscopy of stomach, 276
Diaphragm, action of, in stenosis of upper air-passages, 87
depression of, from emphysema, 173
elevation of, causes displacement of heart, 173
fixation of, in hysteria, 526

Diaphragm, paralysis of, 80, 496
inspiratory dyspnea in, 88
upward displacement of liver from, 289
position of, 71
spasms of, effects of, 496
increased respiration in, 85
Diaphragmatic breathing, 74
friction sounds, 200
phenomenon, 74
Diarrhea, 333
auuria in, 367
brick-dust sediment in urine of, 387
diminution of sweat in, 34
diminished salivation in, 256
from dentition, 254
increase in amount of stools in, 335
infantile, odor of stools in, 336
low specific gravity of urine in, 372
Diastole, movement of blood in, 168
Diastolic collapse of cervical veins, 229
murmur, how distinguished from systolic, 194
in mitral stenosis, 193
Diazo-reaction, 409
Dicrotic pulse, 213
Diet, effect of, on disease, 21
Differentiating electrode, 462
Difficult breathing, 83
Digestion, amylolytic period of, 305
disturbed food-particles in feces in, 347
examination of process of, 304
intestinal, shown by stools, 333
products of, examination of, 321
Digestive apparatus, examination of, 252
disturbances of, in relation to nervous diseases, 526
Dilatation of heart a cause of valvular insufficiency, 168
area of dulness in, 181
eccentric, 169
in anemia, 197
of stomach, reaction of urine in, 372
vomit in, 327
Diminished irritability, significance of, 484
Diphtheria a cause of cyanosis, 38
acute nasal catarrh in, 67
bacillar, differential diagnosis of, from lacunar angina of follicular tonsillitis, 258
bacilli, 551
bacillus, determination of, 260, 262
bacteriological diagnosis of, 260–263
differentiation of, from diphtheroid anginas, 259
from necrotic angina, 259
from scarlet fever, 259
gallop rhythm in, 191
glycosuria in, 402
paleness in, 36
paralysis of tensor of vocal cords in, 543
sometimes no contraction of pupil after, 519
swelling of lymphatic glands of neck in, 260
tone of cough in, 143
tonsils in, 258
Diplococcus in urine, 361
Diplococcus of Fränkel in exudations of empyema, 138
Diplopia, 514
mode of determining, 515
significance of, 515
Direct irritation, 466
Disease, course of, 22
concealment of, 20
exciting causes of, 22
first appearance of, 22
present, 22
previous, as a cause of other disease, 22
simulation of, 20
Disseminated paralysis, reaction of degeneration in, 479
"Dissociated spasms," 425, 490
Distention of stomach, 270, 271
Distoma hæmatobium in blood, 250
in urine, 382
hepaticum, 346
lanceolatum, 346
pulmonale in sputum, 158
Disturbances of speech, 502
of vegetative system in nervous disease, 525
Divergent strabismus, 514
Diverticula of esophagus, sounding for, 267
Dizziness, 438
Dolichocephalus, 431
Dorsal clonus, 460
Double sensibility, 443
vision, 514
significance of, 515
determination of, 515
Drawing-in of abdomen, 278
of chest, inspiratory, 87
Drinks which cause polyuria, 367
Dropsy, anidrosis with, 34
causes of, 47
edema of larynx in, 539
of engorgement with kidney-disease, 399
of kidney-disease, 398
orthopnea with, 29
Drumstick crystals in urine, 389
Dry cough, 143
humming, 128
râles, 128
Duboisin, effect of, on pupil, 517
Duchenne, investigations on motor-points on body, 469
Ductus choledochus, compression of, 302
Duhomme's approximate determination of sugar in urine, 406
Dulness, percussion, 94
relative, 94, 95
heart- and liver-, 108
Dumb-bell crystals in urine, 388
Dumbness, hysterical, 502
Dura mater, diseases of, projections on skull in, 431
Duroziez's double murmur, 222
Dust, inhalation of, a cause of disease, 21
Dynamic sense, testing of, 444
Dynamometer, value of, in testing paralysis of hand, 500
Dysarthria, 502
Dysentery, color of stools in, 337

Dysentery, meat-juice stools of, 338
odor of stools of, 336
pain at stools in, 335
in left iliac fossa in, 278
watery stools in, 338
Dyspepsia, bacteria of mouth increased in, 257
chronic, subacidity combined with retarded evacuation in, 326
coating of tongue in, 255
due to diseased teeth, 253
foul odor of breath in, 252
gastric headache with, 447
headache with, 447
intestinal, yeast fungi in feces in, 349
nervous, 527
hyperacidity in, 325
normal digestion in, 324
vomiting in, 327
watery vomit in, 328
odor from the mouth in, 252
stools in, 337
Dyspnea, 29, 83
alteration of vesicular breathing in, 124
as a factor in nervous disease, 525
color of blood in, 233
expiratory, 88
from emphysema of skin, 50
from uremia, 399
in emphysema of lung, 75
in heart-diseases, 86
in paralysis of diaphragm, 446
inspiratory, 88
lung-, 85
mixed, 89
mydriasis with, 517
objective, 83
of fever, 84
perspiration in, 33
physiological, 83
subjective, 83
Dystrophia, muscular, 455
musculorum, hypertrophy of gastrocnemius in, 456
pseudohypertrophy in, 456

Ear, affections of, in diseases of nervous system, 523
importance of determining disease of, 523
otoscopic examination of, 545
subjective sensibility of hearing in disease of, 523
suppuration of, as a cause of meningitis, 433
Ear-drum, bulging outward of, 546
coalescence of, with wall of tympanum, 546
inspection of, 545
pathological alterations of, 545
retraction of, 545
ruptures and perforations of, 546
Ebstein on lipuria in pyonephrosis, 406
Ebstein's method of determining resistance of heart by percussion, 180
Eccentric dilatation of heart, 169
Ecchymoses, 45
Echinococcus bladders in liver, fluctuation of, 292
Echinococcus in sputum, 157
in urine, 382
in vomit, odor of, 332
membranous rags of, in vomit, 331
of kidney, palpation of, 358
of omentum, 303
of spleen, tumors on surface of organ in, 298
surface of liver in, 292
Eclampsia gravidarum, 490
Eclamptic attacks in children, 490
convulsions, cutaneous hemorrhage in, 46
Ectasia venarum, 46
Edema, 46
cause of, 48
collateral, 48
diseases which cause, 47
distinguished from emphysema of skin, 49
from general venous engorgement, 224
from kidney-disease, 399
from nephritis, 48
in paralysis, 526
inflammatory, 48
malignant, bacilli of, 550
of chest-wall, deadening of chest-resonance in, 112
diminished vesicular breathing in, 124
of larynx, appearance of, 538
of lungs, crepitant râles in, 131
dyspnea in, 85
hemorrhage in, 146
non-uniform crepitation in, 132
serous sputum in, 145
tympanitic sound in, 98, 113
with pneumonia, prune-juice sputum in, 145
of skin and subcutaneous cellular tissue, 46
weakened apex-beat in, 174
Effusion in abdominal cavity, 282
into pleural cavity, change of boundaries of deadness in, 112
of blood, reaction of urine in, 372
Egophony, 135
Egyptian chlorosis, 345. See *Anchylostomiasis*.
paleness in, 36
Ehrlich, gigantoblasts, 240
Ehrlich's diazo-reaction, 409
method of staining tubercle bacilli, 160
Eichhorst on Ebstein's percussion for determining resistance of heart, 180
on odor of echinococcus in vomit, 332
on paint-like odor of sputum, 147
on the sound of crepitant râles, 131
Einhorn's apparatus for illuminating stomach, 276
fermentation saccharometer, 404
Elastic fibers, how to obtain for examination, 153
in sputum, 152
Electric sensibility to pain, 441
stimulation of nerves, 467
Electrical condition, diagnostic value of, 484
examination in detail, 469
of nerves and muscles, 462
reaction, mixed, 483
Electrode, differentiating, 462

Electrode, Erb's fine, 463
use of, 473
indifferent, 467
Emaciation, how detected, 31, 32
significance of, 31
Emboli, effect of, on pulse, 211
of central retinal artery a precursor of cerebral embolism, 547
Embolism, local, low temperature in, 65
Embolus of central artery of retina, 548
of retinal artery in endocarditis aortæ, 514
Embryocardia, 190
Emesis, induction of, when contraindicated, 304
Emphysema a cause of hypertrophy of right ventricle, 120
anomalies of breathing in, 80
anuria in, 368
aspiration, 49
diminution of expiratory pressure in, 141
distinguished from edema, 49
downward displacement of liver in, 289
dry râles in, 129
dyspnea in, 75
enlargement of liver in, 289
gallop rhythm in, 191
liver-dulness in, 295
of lung, abnormally loud percussion-sound in, 116
a cause of cyanosis, 39
a cause of displacement of apex-beat, 173
band-box note in, 116
diminished vesicular breathing in, 124
diminution of heart-dulness in, 181
of spleen-dulness in, 301
dyspnea in, 85
expiratory dyspnea in, 88
extension of boundaries of lungs in, 117
from bronchial asthma, 89
non-tympanitic sound in, 99
râles with, 128
systolic pulsation in epigastrium, 177
thorax in, 75
vein-nets on chest and back in, 225
venous engorgement in, 224
vicarious, one-sided downward movement of lung-boundary in, 117
of mediastinum, diminished heart-dulness in, 182
of skin, 49
diagnostic importance of, 50
weakened apex-beat in, 174
peptonuria with, 398
prolonged expiration-sound in, 124
relative liver-dulness in, 296
vicarious, 76
Emphysematous thorax, 75
Emprosthotonos, how produced, 496
Empyema, amount of expectoration in, 144
bacteria in exudations of, 138
expansion of chest in, 77
Fränkel's pneumococcus in pus of, 163
hematoidin-crystals in pus of, 156
in sputum in, 147
necessitatis, 90
pulsans, 90, 176
Empyema, pulsatile, 90
pulsation in heart-region in, 176
purulent sputum in, 145
staphylococcus pyogenes in, 548
tyrosin in sputum of, 157
Encapsulated pleurisy, 111
Encephalitis, arrest of growth of bones after, 530
chronic motions in, 492
epileptiform attacks in beginning of, 490
neuroretinitis in, 547
Endocardial murmurs distinguished from pericardial, 199
loudness of, 195
whizzing, 196
Endocarditis a cause of insufficiency, 168
a cause of stenosis of a valve, 168
acute, hematuria in, 375
aortæ, embolus of retinal artery with, 514
embolus of central artery of retina in, 548
hematuria with, 375
mitralis, embolus of retinal artery in, 514
pus-cocci in urine in, 385
streptococcus pyogenes in, 549
ulcerative, staphylococcus pyogenes in, 548
Engorgement, abnormal redness of mucous membrane of larynx in, 538
of kidneys, albuminuria in, 394
hematuria from, 375
of liver, surface of liver in, 291
of spleen, tenderness in, 298
Enlarged glands, pressure by, upon recurrent nerves, 543
Enrichment defined, 553
Enteritis, color of stools in, 337
poor resorption of fats in, 335
Enteroliths in stools, 340
Enteroptosis, floating kidney a symptom of, 358
Enuresis from irritation of penis, 529
nocturna, 528
Eosinophile cells, 242
Epigastric angle, 73
Epigastrium defined, 268
illustrated, 269
pulsation of, 177
Epilepsy, acetonuria after, 409
albuminuria after, 394
coma from, 437
convulsions or spasms of, 490
cyanosis in, 39
dyspnea in, 95
glycosuria after, 402
hemorrhage into subcutaneous tissue in, 530
involuntary discharge of urine in, 528
Jackson's, 490
partial, 490
polyuria in, 367
punctiform ecchymoses after, 529
spermatozoa in urine in, 379
spermatorrhea in, 414
Epileptic attacks, fever in, 525
tonic spasms in, 490
wounds of tongue from biting in, 254
Epileptiform attacks in children, 490
Epistaxis, 66

Epithelial casts in urine, 382
cells in saliva, 256
in sputum, 151
in stomach-contents, 316
Epithelium in sputum, 151
in stools, 348
in urine, 378
in vomit, 331
Equinia, acute nasal catarrh in, 68
bacilli in urine, 385
Erb, muscular dystrophia, 455
myotonic reaction, 483
on partial reaction of degeneration, 483
Erb's electrode for testing paradocutaneous sensibility, 442
fine electrode, 463
use of, 473
point, 471
Erector trunci, paralysis of, 495
Ergotism, symmetrical gangrene in, 526
Ernst on nature of hyaline casts, 381
Eructation, 327
Erysipelas cocci in urine, 385
continual fever in, 59
enlargement of spleen in, 298
leukocytosis in, 242
staphylococcus pyogenes in, 548
vomiting in, 326
Erythema exudativum multiforme, 44
Erythrodextrin from digestion of starch, 304
test for, 321, 322
Esbach's albuminometer, 397
Eserin, effect of, on pupil, 517
Esophagoscopy, 268
Esophagus, anatomy of, 263
auscultation of, 268
dilatation of, 267
diverticula of, detection by sounding, 267
examination of, 263
of neighborhood of, 267
with sound, 264
obstruction of, 267
palpation of, 264
percussion of, 267
sounding of, difficulties and dangers of, 265
stenosis of, characteristic distress with, 263
stricture of, location of, by sounding, 266
Esthesiometer, use of, 440
Ether-poisoning, jaundice from, 42
Etiological diagnosis, 17
Ewald's salol method of determining rapidity of evacuation of stomach-contents into duodenum, 322
test-meal, 311
Examination, chemical, of aspirated fluid, 139
electrical, 462–485
of arteries, 201
of blood, 231
of digestive apparatus, 252–354
of œsophagus, 263
of feces, 332
of heart, 166–201
of intestines, 277
of kidneys, 355
of lips, 253
of liver, 286–296
of mouth, 252
Examination of nervous system, 415
of omentum, 302
of pancreas, 302
of patient, 24
form for recording, 26, 27
general, 28
of peritoneum, 281
of secretions of male genito-urinary apparatus, 411
of stomach, 268
of stomach-contents, 303
of spleen, 296
of teeth, 253
of tongue, 254
of urinary apparatus, 355
of urine, 360
of veins, 223
Examining electrode, application of, 467
Exanthemata from poisons and use of medicines, 45
Excitability, mechanical, of muscles and nerves, 485
quantitative, of nerves and muscles, 476
Excitement, effect of, on amount of urine, 636
headache after, 446
strengthening of heart-sounds in, 187
syncope in, 438
Exophthalmos paralyticus, 515
Expectoration, 143
examination of, 144
general characteristics of, 144
green, 147
hemorrhage with, 146
odor of, 147
varieties of, 144
Expiration, auxiliary muscles of, 87
Expiratory bulging, 88
dyspnea, 88
Exploratory puncture of abdomen, 285
of heart, 200
of pleura, 136
Explosive speech, 502
Extensors of hand, paralysis of, 498, 499
Extirpation of pancreas a cause of diminished fat-digestion, 348
Extra-pericardial friction-sounds, 200
Extravasations, chylous and chyliform, from the pleural cavity, 137
Extremity, measurement of volume of, 453
Exudation, diagnosis from transudation by exploratory puncture, 137
Exudations, examination of, for bacteria, 138
in cavum tympani, 546
metapneumonic, 138
microscopic appearances of, 137
plegaphonia over, 135
Eye, affections of, in diseases of nervous system, 513
cysticerci in, 527
movements of, 514
paralysis of muscles of, 513–515
significance of, 516
Eyelids, dropsy of, from kidney-disease, 398

Face, motor-center for lower part of, 417
muscles of, 492, 493
Facial nerve, effect of paralysis of, 493

Facial nerve, lesion of, from caries of petrous bone, 436
motor tracts of, illustrated, 426
paralysis, 493
cerebral, 493
diminished secretion in, 527
hearing in, 523
testing of taste in, 524
phenomenon, 485
reflexes, 460
tract, paralysis of, bone-reflex in, 461
Facialis, course of, 418
Faradic apparatus for electrical examination, 462
current causes tetanic contraction, 467
examination of nerve, method of, 473
Faradocutaneous sensibility, 441
Fat-drops in urine, 378
development of, in different persons, 30
in conjunctiva not to be confounded with jaundice, 40
in stools, 348
in urine in health, 366
make it turbid, 371
variations in amount of, 30
Fatigue as a factor in causation of disease, 21
Fatty-acid crystals in sputum, 156
in sputum of gangrene of lungs, 164
in stools, 348
in urine, 407
degeneration of heart, Cheyne-Stokes respiration in, 82
of kidney, renal epithelia in urine of, 379
heart, paleness in, 37
retardation of pulse in, 204
stools, 338
Favus fungus in vomit, 332
Febrile diseases, ascending venous pulse in, 229
diminished salivation in, 256
heart-murmurs in, 197
retardation of pulse in, 204
with heart disease, frequent pulse in, 205
hyperemia of mucous membranes, redness of mucous membrane of larynx in, 538
Febris hepatica, herpes with, 45
Fecal accumulation in stenosis of intestine, 334
odor of stools, 336
vomiting, 330
Feces, amount of, 335
balls of, in intestine, palpation of, 279
chemical examination of, 353
examination of, 332
microscopic examination of, 347
mixed with blood, significance of, 339
physical and chemical properties of, 336
Feculent exudation in pleural exudation, 139
odor of urine, 373
Feeling of constriction, 446
Fehleisen on erysipelas cocci in urine, 385
Fehling's solution in testing for sugar in urine, 404
Female sexual organs, headache with disease of, 447
Femoral vein, compression or thrombosis of, engorgement of cutaneous veins of leg from, 224
Ferment in sputum, 164
Fermentation of stomach-contents, 309
of urine, alkaline, a sign of cystitis, 383
caused by micro-organisms, 383
reaction of urine in, 373
ammoniacal, odor of urine in, 373
test for sugar in urine, 403
Ferments, function of, in digestion, 306, 307
Fever, 50, 54
acetonuria in, 409
albuminuria in, 394
anuria in, 367
as a symptom in nervous diseases, 531
brick-dust sediment in urine in, 387
casts in, 380
chill of, paleness in, 36
chlorid of sodium in urine diminished in, 392
coating of tongue in, 255
color of urine in, 369
continued. See *Continued fever.*
daily difference of, 56
diaceturia in, 407
diminished alkalescence of blood in, 250
dryness of tongue and throat in, 254
exacerbation of, 56
frequent pulse in, 204
hectic. See *Hectic fever.*
hyaline casts in urine of, 380
hyperpyretic, 55
importance of observing pulse in, 205
increased mucus in urine of, 375
increase of urea in, 392
increased frequency of respiration in, 84
in nervous diseases, 525
intermittent. See *Intermittent fever.*
irregular, 64
lips in, 253
paroxysm, 63
perspiration in, 33
pulse in, 204
recurrent. See *Recurrent fever.*
red skin in, 37
remission of, 56
remittent. See *Remittent fever.*
sounds over arteries in, 222
symptom-complex of, 54
three types of, 56
thrush in, 255
typical courses of, 59
typus inversus of, 56
uric acid increased in, 392
urobilinuria in, 369
variations of temperature in, 56, 57
with albuminuria, 399
Fibrillary contractions, 489
Fibrin in cerebrospinal fluid, 435
in urine, 398
Fibrinous tubes in sputum, 148
Fibroma of larynx, 541
Field of vision, concentric narrowing of, 521
testing of, 520
Filaria sanguinis hominis, effects upon urine and urinary passages, 382

Filaria sanguinis hominis in blood, 250
Filehne on ferment in sputum, 164
Filiform pulse, 209
Finger-percussion, 93
Fingers, paralysis of muscles of, 498
Fischer, polycsthesia, 443
Fischl on origin of epithelium in sputum, 151
Fissure of anus, pain at stools with, 335
of Rolando, location of, 432
of Sylvius, location of, 432
Fleiner's modification of Mietz's test for free hydrochloric acid, 320
test-meal, 312
Fleischl's hemometer, 234
Flexibilitas cerea, 492
Flimmering, 522
Floating kidney, 358
Fluctuation a sign of fluid, 283
Flügge on bacilli of malignant edema, 550
Fluid in pericardium, effect on heart-dulness, 181
in peritoneal cavity, 283
palpation and percussion of, 283, 284
Fœtor ex ore, 252
Folie musculaire, 492
Food digested in feces, 347
effects of, upon color of stools, 336, 337
undigested, in stools, 336, 337, 347
-value of nourishment, determination of, 353
Foot-clonus, 460
paralysis of muscles of, 501
perforating disease of, 530
-phenomenon, 460
Forearm, paralysis of muscles of, 498
Fossa infraspinata, 69
subspinata, 69
supraclavicularis, 68
Francke's scarificator, 232
Fränkel's pneumococcus in sputum, 162
staining of, 163
Frédéricq-Thompson on red border on gums in tuberculosis, 254
Frémissement cataire, 196
Fremitus, laryngeal, 67
vocal, palpation of, 133
Frerichs on artificial distention of stomach, 271
on cause of icterus neonatorum, 42
"square position" of liver, 295
Frick on cause of green sputum, 147
Friction-sounds, extra-pericardial, 200
palpable, 132
pericardial, 198
pleuritic, 132, 200
Friedländer's pneumococcus in sputum, 162
staining of, 163
Friedreich on "change of respiratory sound," 99
on diastolic collapse of cervical veins, 229
on expiratory valvular sound in crural vein, 230
on starch-corpuscles in sputum of hemorrhage from lungs, 155
on venous sound in tricuspid insufficiency, 230
Friedreich's respiratory change of sound, 115
Fruity odor of urine, 373
Fuliginous deposit on lips, 253
Fulness of veins, increased, 223
Fundus oculi, changes in, in nervous diseases, 546
changes in, in other than nervous diseases, 547
of stomach, position of, 269
Fungi in sputum, 158
in urine, 383
pathogenic, in stools, 349
sprouting, in stomach-contents, 316
Fungus-spores in feces, 349
Funnel-breast, 79
Fürbringer on oxalate of lime in sputum of diabetes, 157
on pear-concretions in stools, 340
on seminal fluid, 411
on temporary relative aspermatism, 412
urethrorrhœa ex libidine, 413
Fürbringer's reaction, 396
Furuncles, staphylococcus pyogenes in, 548

Gabett's method of staining tubercle bacilli, 161
Gabritschewsky's pneumatoscope, 136
Galacturia, 371
caused by filaria sanguinis hominis, 383
Gall-bladder, enlargement of, liver dulness in, 296
location of, 287
normal and pathological condition of, 292
obstruction of, enlargement of liver in, 289
palpation of, 292
Gall-stones, appearance of, described, 340
chemical tests for, 340
fever in, 57
in feces, how to find, 340
in stools, 339
diagnosis of, from pear-concretions, 340
palpation of, 292
Gallop-rhythm, 190
Galvanic apparatus for electrical examination, 462
current, effect of, only at closing and opening of current, 467
how to distinguish the poles, 416
measurement of, 465
quality of reaction with, 467
examination of nerve, method of, 475
resistance, testing of, 442
Galvanometer, absolute, 465
Edelmann's, 478
Gangrene, infusoria in sputum of, 158
"odorless," 147
of lungs, crystals of fatty acid in sputum of, 156
elastic fibers in sputum of, 152, 153
fatty acids in sputum of, 164
ferment in sputum of, 164
hematoidin-crystals in sputum of, 156
mucopurulent sputum in, 145
odor of sputum in, 147
starch-corpuscles in sputum of, 155
spontaneous symmetrical, in nervous diseases, 526

Gangrene, starch-corpuscles in sputum of, 155
Gärtner on bacillus enteritidis, 554
Gases in stomach-contents, 310
Gastric crises, 527
 crisis of tabes, hypersecretion of gastric juice in, 325
 juice, action of, 305
 bactericidal action of, 305
 determination of digestive power of, 321
 hypersecretion of, 311
Gastritis, phlegmonous, vomiting of pus in, 330
Gastrodiaphanoscopy, 276
Gastroduodenal catarrh a cause of jaundice, 41
Gastrointestinal disturbance, acetonuria in, 409
Gastroxynsis acuta, hyperacidity in, 325
 superacidity in, 325
Geigel on heart-sounds, 183 (note)
 on systolic pulse in vena cava, 229
Geisler's albumin test-papers, 395
General examination of patient, 28
Genital apparatus, disturbances of, relation to nervous disturbances, 529
Genito-urinary secretions, male, examination of, 411
Gerhardt, chlorid-of-iron reaction of urine, 373
 on displacement of lower border of lung in lying down, 109
 on interrupted Wintrich's change of sound, 114
 on urobilin-icterus, 42
 reference to, as improver of percussion, 92
 sound over crural artery in pregnancy, 222
 (D.) on bilirubin in blood in icterus, 43
Gerhardt's change of sound, 115
 chlorid-of-iron reaction for diacetic acid, 407
Gerlach on artificially-produced Curschmann's spirals, 155
Gigantoblasts, 240
Glanders-bacillus in blood, 247
Gleet may be due to tuberculosis, 384
Glossitis, parenchymatous, enlargement of tongue in, 254
Glossy skin, nervous origin of, 529
Glottis, paralysis of dilators of, dyspnea with, 88
 spasm of, 541
 vibration of, in phonation, 133
Gluteus maximus, paralysis of, effect of, 500
Glycosuria, 401
 alimentary, 366, 402
 diagnosis between physiological and pathological, 402
 with diseases of oblongata, 528
Gmelin's test for bile-pigments in urine, 400
Goldschneider on perception of movement, 445
Goldschneider's movement measure, 445
Gollash on eosinophilous asthma-cells, 157
Gonococci in pus of gonorrhea, 384
Gonococcus, 549
Gonorrhea, hematuria from, 376
 gonococci in urine of, 384
 threads of mucus in urine of, 375
Gonorrheal threads in urine, 375
"Goose's throat," 220
Görges on physiological variation in reaction of urine, 364
Gottstein on ulcers characteristic of tuberculosis of larynx, 540
Gout, diabetes in, 447
 neuralgia in, 447
 uric acid in blood in, 250
Gouty diathesis, uric acid increased in, 392
Gowers's hemoglobinometer, 234
Granular hyaline casts, 381
Grape-sugar in urine, 401
Graphic communication, disturbance of, 503
 representation of aphasia, 509–513
Gray skin of silver deposit, 44
Green sputum, 147
Griesinger on anchylostomiasis, 345
Growth of hair in nervous disease, 530
Guaiac-tincture test for bile-pigment in urine, 400
Gummata of larynx, 540
 syphilitic, of skull, projections caused by, 431
 on surface of liver, 292
Gumprecht's method of percussing heart, 180
Gums, examination of, 253
Güterbock on temporary relative aspermatism, 412
Gutbrod, recoil-theory of apex-beat, 172

HABITATION, influence on disease, 21
Habits, influence of, on disease, 21
Habitual constipation in nervous disease, 527
Hæmatobium malariæ in blood, 247
Haeser on specific gravity of urine as a guide to amount of solid constituents, 372
Hair, growth of, dependent on neurosis, 530
Halfmoon-shaped space, diminution in size of, 275, 276
 of, by enlargement of spleen, 302
 of Traube, 270, 275
Hammer-pleximeter percussion, 93
Hammond (W. A.), athetosis, 492
Hand and fingers, characteristic positions of, in paralysis, 499
 distribution of nerves of, 449
 examination of, in paralysis, 499
 paralysis of muscles of, 498, 499
Hauser on sarcina pulmonalis, 159
Hawking, 143
Hayem on urobilinuria in liver-affections, 369
Hayem's counting-chamber, 239
 fluid, 239
Head, distribution of nerves of, 448
Headache, 446
 anemic, 447
 from uremia, 399
 habitual, 447
 toxic, 447
Hearing, center for, 421
 subjective sensibility of, 523
 testing of, 522
 tract of, 420
Heart-action, rapidity and strength of, 183
 anatomy of, 166
 auscultation of, 182

Heart-beat, broad, 176
rhythm of, 183
change of form of, 170
dilatation of, retardation of pulse in, 204
tympanitic sound in, 112
diminished work of, weakens apex-beat, 174
-disease, anuria in, 368
cells in sputum in, 151
Cheyne-Stokes' breathing in, 82
cyclic albuminuria in, 396
dyspnea in, 86
edema in, 48
pulse in, 207
venous engorgement in, 224
dislocation of, a cause of displacement of apex-beat, 172
-dulness, absolute, 177
diminution or loss of, 181
displacement of, 182
enlargement of area of, 180
in children, 178
relative, 108, 177, 179
in children, 180
size and diagnostic value of, 179
enlargement of, a cause of displacement of apex-beat, 173
expansion of chest in, 77
simulated, 171
examination of, 166–201
exploratory puncture of, 200
-failure, frequent pulse in, 207
fatty degeneration of, Cheyne-Stokes' respiration in, 82
hypertrophy of. See *Hypertrophy*.
compensatory, 169
other than valvular, 170
tympanitic resonance in, 112
inspection and palpation of, 171
left, enlargement of, smallness of Traube's space in, 275
-liver boundary, 294
movement of blood in, 167
-murmur, presystolic, 194
systolic, heard over carotid, 222
-murmurs, anemic, 197
combination of several, 196
differentiation of, 196, 197
endocardial, loudness of, 195
inorganic, 197
localization of, 192
organic, 191
organic endocardial, 191
relation of, to time of heart's action, 192
transmission of, 196
neuroses, pain in, 526
percussion of, 177
physiology of, 167
position of, in thorax, 166
prominence in neighborhood of, 175
pulsation in region about, 176
pulsations at base of, 176
palpation for, 90
relation between size of, and parietal portion, 170
relation of, to nervous disease, 526
simple dilatation of, 170
Heart-sounds, divided, 190
double, 190
metallic, 191
normal, 183–187
variations in, 186
pathological changes in, 187
rhythm of, alteration in, 189
strengthening of, 187
tone of, variations of, 187
tones of, 183
weakness of, 189
Heat and cold, sense of, testing of, 440
sensations of, 526
"Heaving" of apex-beat, 174
Hectic fever, 61
redness in tuberculosis, 37
Hegar on injection of water in palpation of rectum, 280
Heger-Kaltenbach, palpation of ureters through vagina, 359
Heller's test for blood in urine, 400
for hemin, 330
Hematemesis, 328
Hematin in solution, test for, 399
Hematochyluria, 250
Hematoidin-crystals in sputum, 147, 155
in urine, 389
Hematoma of dura, inequality of pupils in, 517
Hematuria, 375
albumin with, 399
blood-casts a sign of hematuria, 382
caused by filaria sanguinis hominis, 383
coloring-matter of blood in urine in, 370
due to strongylus gigas, 382
fibrin in urine of, 398
when occurs, 375
Hemialbumose in urine, 394, 398
Hemianesthesia, hysterical, unilateral paleness in, 526
in gross hysteria, 491
when occurs, 443
Hemianopsia, 521
homonymous, 521
how produced, 421
light-reaction of pupil in, 519
schematic representation of, 520
Hemianoptic pupil rigidity, 519
Hemiathetosis, 492
Hemichorea, 492
Hemicrania, unilateral paleness or redness in, 526
Hemidrosis, 33, 34
Hemin in urine, test for, 400
-test for blood in vomit, 330
Hemiopia, how produced, 421
Hemiplegia alterans, 453
cruciata, 453
seu alterans, 425
decubitus acutus in, 530
defined, 452
deforming arthritis in, 531
dorsal flexion of foot in, 492
due to lesion of internal capsule, 425
stiffness of joints in, 531
Hemisystole, double, positive venous pulse in, 229

Hemisystolia, 175
Hemoglobin, alteration of percentage of, in anemia and chlorosis, 238
casts of, in urine, 382
in blood, determination of amount of, 233
in urine, appearance of, 377
tests for, 399
Hemoglobinemia, coloring-matter of blood in, 370
recognition of, 233
spectroscopic examination of blood in, 235
Hemoglobinometer of Gowers, 234
Hemoglobinuria, albumin with, 399
appearance of urine in, 377
coloring-matter of blood in urine in, 370
paroxysmal, 371
Hemo-hepatogenous icterus, 41
Hemometer of Fleischl, 234
Hemophilia, hematuria in, 376
Hemoptysis, 146
Hemorrhage, amaurosis after, 548
cystic, appearance of blood in urine from, 376
from intestine, bloody stools in, 338
from lungs, 146
starch-corpuscles in sputum of, 155
from stomach, blood mixed with feces in, 339
location of source of, in hematuria, 376
microcythemia after, 240
of kidney, spirillum recurrens in urine of, 385
of lungs, diagnosis of, 146
of pons or oblongata, effect of, 425
of skin in hysteria, 529
of stomach, bloody vomit in, 329
odor of vomit in, 332
retardation of pulse in, 204
retinal, 547, 548
renal, appearance of blood in urine of, 376
subnormal temperature with, 57
symptoms of, 36
Hemorrhages, cutaneous, 45
disturbance of consciousness from, 437
into subcutaneous tissue in epilepsy, 530
in the skin, 45
in the skin, distinguished from inflammation, 45
Hemorrhoids, hemorrhage from large intestine in, 338
pain at stools with, 335
Hepatitis, diffuse, enlargement of liver in, 289
interstitial, surface of liver in, 291
Hepatization of lung, resonance over, 109
Hepatogenous icterus, 40
Hereditary disease, 20
Heredity, 20
Hering's spectroscope, 235
Herpes facialis, 44
labialis, 45
nasalis, 45
nervous origin of, 529
zoster, when occurs, 529
Herringham on cyclic albuminuria, 396 (note)
Heubner on detection of metallic percussion-sound, 99
rod-plexímeter, percussion of, 117
Hiccough, 496
High percussion-sound, 95
Hippocratic succussion, 133
Hissing râles, significance of, 128
History, clinical, defined, 17
Hoffmann (F. A.), on hemosiderin, 151
Hoffmann's test for perception of form, 446
Hofmeier on cause of icterus neonatorum, 42
Holz on ascending venous pulse, 229
on cultures of bacillus of typhoid fever, 550
Homonymous hemianopsia, 521
Hooke, reference to, 118
Hoppe-Seyler on fatty acids in sputum of gangrene of lungs, 164
Horseshoe kidney, 356
Hour-glass crystals in urine, 388
stomach, recognition of, by inflation, 272
Huber on unreliability of Ewald's salol method, 322
Hübner, congo-red test for hydrochloric acid, 316
Humming sounds, significance of, 128
venous, 230
Hürthle on the "recoil" of the pulse-curve, 212
Hutchinson's spirometer, 141
teeth, 253
triad, 253
Hyaline casts, 380
chemical nature and origin of, 381
granular, 381
staining of, 380
Hydatid thrill, 292
vibration with echinococcus of kidney, 358
Hydremia, 36
albuminuria with, 394
edema in, 47
leukocytosis in, 242
polyuria in, 366
Hydrobilirubin in urine, 42
Hydrocephalus, anosmia in, 524
choked disk in, 547
enlargement of cranium in, 430
falling forward of head in, from weakened or paralyzed muscles, 495
how distinguished from rachitic, 431
Hydrochloric acid, antiseptic and peptic power in digestion, 307
diminished secretion of, effect on digestion, 308
in stomach, deficiency of, determination of, 320
determination of, 316
tests for, 316–321
value of, 318
increased secretion of, 309
masked, 306
of stomach, function of, 305
period of digestion, 306
Hydronephrosis, palpation of, 358
temporary flow of urine after, 367
tenderness with, 357
Hydropericardium, area of heart-dulness in, 184
weakened heart-sounds in, 189
Hydropneumothorax, exploratory puncture in, 136

Hydropneumothorax, splashing in, 133
Hydrops mechanicus, 47
vesicæ fellcæ, 292
Hydrothionic urine, 373
Hydrothorax, boundaries of percussion-dead-ness in, 111
exploratory puncture in, 136
Hypacidity, microscopic appearance of stomach-contents in, 316
of stomach, significance of, 324
varying significance of, 324
Hyperacidity, microscopic appearance of stomach-contents in, 316
of stomach, 308, 309
relative, 310
significance of, 325
Hyperesthesia, 443
ovarian, 529
Hyperidrosis, 33
localis, 33
universalis, 33
Hypermetropia, inequality of pupils in, 517
Hyperosmia, 524
Hyperpyretic fever, 55
Hyperpyrexia, 55
Hypersecretion of gastric juice, 311
significance of, 325
vomit in, 328
of stomach, acid gastric juice after test-breakfast, 323
Hypertrophic liver, surface of, 291
Hypertrophy of heart, area of heart-dulness, 180
compensatory, 169
other than valvular, 170
pulse in, 210
of left ventricle favors hemorrhage and valvular endocarditis, 526
from nephritis, 170
from sclerosis of arteries, 170
idiopathic, 170
large pulse in, 208
pulsation of aorta in, 218
of muscles, 453, 456
of right ventricle from emphysema, 170
strengthened pulmonary sound in, 188
of stomach, peristaltic motions in, 272
Hypesthesia, 442
Hyphidrosis, 33
Hypnosis, catalepsy in, 492
Hypochondria, oxalate of lime in urine of, 389
phosphaturia in, 389
Hypochondrium defined, 268
illustrated, 269
Hypogastrium defined, 268
illustrated, 269
right, in enlargement of liver, 288
Hypoglossus, course of, 418
motor center for, 416
peripheral paralysis of, speech disturbance in, 502
tract, 418
Hysteria, active spasms in, 456
ageusis in, 524
allochiria in, 443
anesthesia of pharynx in, 527
Hysteria, anuria in, 528
catalepsy in, 492
central vomiting in, 326
concentric narrowing of field of vision in, 521
dyspnea in, 526
epithelial cells in bloody sputum of, 151
gross, 491
headache in, 447
hemianesthesia in, 443
hemorrhage of skin in, 529
hypersecretion of gastric juice in, 325
importance of disease of female genitals as a cause of, 529
increased tendon-reflexes in, 461
laryngeal paralysis in, 525
loss of voice in, 495
nervous cough in, 525
oliguria in, 528
palpitation and pain of heart in, 526
paralysis of larynx in, 68
of laryngeal muscles in, 541
of thyreo-arytænoideus in, 542
polyuria in, 528
raspberry sputum in, 146
retardation of pulse in, 204
sensibility of cranium to pressure, 433
sensitiveness of spine to pressure in, 434
severe, 491
tonic spasms in, 490
tremor with, 489
urinary disturbances in, 528
vasomotor disturbauces in, 526
vomiting with, 527
Hysterical convulsions, 491
dumbness, 502
hemianesthesia, unilateral paleness in, 526
Hystero-epilepsy, 491
cyanosis in, 39
Hysterogenous zones, 529
in gross hysteria, 491
Hystero-traumatic neuroses, analgesia in, 444

Icteric casts in urine, 380
urine, 371
Icterus, 40. See also *Jaundice.*
bile-pigment in urine of, 371
in sputum in, 147
casts in urine in, 380
catarrhal, oxalate of lime in urine of, 381
color of urine in, 371
from dissolution of blood by poisons, 41
hemo-hepatogenous, 41
hepatogenous, 40
color of stools in, 337
retardation of pulse in, 204
neonatorum, 42
of stagnation, 40
urobilin, 42
Idiocy, 438
Idiomuscular contractions, 485
Idiopathic migraine, 447
neuralgia, 447
Idiotism, increased secretion of saliva in, 527
Ilco-psoas, paralysis of, effect of, 500
Ileus, 330
odor of vomit in, 332

Iliac fossa, right, pain on pressure over, 277
Imagination, examination of, 437
Imbecility, 438
Inactivity, atrophy from, 453
Inanition, acetone in urine in, 409
retardation of pulse in, 204
Incisura cardiaca and parietal relation of heart, 171
boundaries of lungs at, 106
Incompensation of heart, 189
Incontinentia alvi, 335
in nervous disease, 527
urinæ, 528
Increased irritability, significance of, 485
Incubation period defined, 22
Indican, abnormal amount in urine, 369
in urine, test for, 369
of urine, 363
Indicanuria, 369
Indifferent electrode, 462
method of using, 467
Indirect irritation, 466
Individual diagnosis, 18
Indoxylsulphuric acid in urine, 409
Infantile paralysis, muscular hypertrophy in, 456
Infarction, hemorrhagic, Curschmann's spirals in, 155
of kidney, hematuria in, 375, 376
intestinal, 338
of lungs, bronchial breathing in, 125
friction-sounds in, 133
hemorrhage in, 146
percussion-sound in, 101
of spleen, enlargement of organ in, 298
tenderness in, 298
pulmonary, crepitant râles in, 131
Infectious diseases, 20
acute, delirium in, 438
disturbance of consciousness in, 437
headache with, 447
breathing in, 81
Cheyne-Stokes' respiration in, 83
enlargement of liver in, 289
fever in, 57
glycosuria in, 403
hemoglobinuria in, 370
tenderness of spleen in, 298
influence, neuralgia from, 447
Inflated thorax, 75
Influenza bacillus, 552
Infrascapular space, 69
Infraspinatus muscle, function of, 498
Infratrochlear nerve illustrated, 448
Infusoria in feces, 353
in sputum, 158
Inguinal region defined, 268
illustrated, 269
Inherited diseases, 20
tendency, 20
Inorganic murmurs, 197
sediments in urine, 385
Inosite in urine, 406
Insanity, supercidity in, 325
spermatorrhea, 414
Inspiration, auxiliary muscles of, 86
Inspiratory dyspnea. See *Dyspnea.*
Insufficiency, aortic, character of murmurs in, 195
diastolic murmur in, 193
increased pulsation in arteries, 221
liver-pulse in, 228
pathological sound over arteries in, 22
pulsating tumor of spleen in, 298
pulsation of aorta in, 208
pulse-curve in, 214, 215
quick pulse in, 209
weakening of first sound at apex in, 189
caused by endocarditis, 168
mitral, division of second apex-sound in, 190
murmurs in, character of, 195
pulse in, 216
systolic murmur in, 193
murmur of, 192
of aortic valve, effects of, 168, 169
of heart valves, murmurs in, 191
of mitral valve, effect of, 168, 169
of pulmonary valve, effects of, 168, 169
of stomach, determination of, by test-meal, 314
food in stomach after digestion of test-breakfast, 323
of tricuspid valve, effect of, 168, 169
pulmonary, diastolic murmur in, 193
pulse in, 217
relative valvular, 168
tricuspid, positive venous pulse in, 227
systolic murmur in, 193
venous engorgement in, 224
venous sound in, 230
valvular, and its effects, 168
Intention tremor, 489
Intercostal neuralgia, 90
in tabes, 447
spaces, systolic drawing in of, 177
Intermittent fever, 63
chlorid of sodium in urine increased in, 392
herpetic eruption in, 45
in febrile diseases, 63
malarial, 63
range of temperature in, 56
sweat in, 34
Internal capsule, blood-supply of, 422
effect of lesion of, 425
lesions of, hemianesthesia in, 443
hemorrhage, 36
Interscapular space, 69
Intestinal catarrh. See *Catarrh.*
contents, accumulation of, indicanuria in, 369
crises, 527
digestion shown by stools, 333
discharges, pregnancy and character of, 333
infarction, 338
parasites as a cause of nervous diseases, 527
peristalsis, visible, 279
Intestine, auscultation of, 281
exploratory puncture of, 285, 286
occlusion of, indicanuria in, 369
percussion of, 280
deep, 281

Intestine, small, mistaken for colon, 279
tumors of, palpation of, 279
percussion of, 280
Intestines, examination of, 277
metallic sound over, 98
palpation of, 277
tenderness of, 277
Intoxications, retardation of pulse in, 204
Intussusception of intestine, obstipation in, 334
Invagination of intestine, obstipation in, 334
Iodid of potassium in saliva, 256
Iodin in urine, test for, 410
Iron dust in sputum, 147
Irrigation of rectum, 286
Irritability, diminished, significance of, 484
increased, significance of, 485
qualitative, of muscles, 478
Ischiatic nerve, electric stimulation of, 472
Ischuria paradoxa, 528
Itching in jaundice, 41

JACKSON'S epilepsy, 490
Jaffé on fatty acids in sputum of gangrene of lungs, 164
Jaquet's sphygmograph, 211
Jaundice, 40. See *Icterus.*
caused by ascaris lumbricoides, 343
from gall-stones, 339
itching in, 41
sweat in, 34
Jendrassik on examination of foot-phenomenon, 461
Johann Faber's dermatograph, value of, 133
Johnson on picric-acid test for albumin in urine, 395
Johnson's modification of Widal test, 247
Joint-disease in diseases of nervous system, 530
neuroses, 531
Joints, chronic diseases of, atrophy with, 455
Jolly on contraction of myotonic muscles, 484
Jonás and Benczúr. See *Benczúr and Jonás.*
Jugular vein, abnormal fulness of, 223, 224
phenomena of circulation in, 225
valvular sound in, 227
Jurasz on "cerebral blowing," 222

KAHLER on hemialbumose in urine, 398
Kannenberg on spirillum recurrens in urine, 385
Karlinski on bacillus enteritidis, 554
Kast on effect of absence of hydrochloric acid from gastric juice, 325
on fermentation-test for sugar, 404
Kidney, amyloid, hyaline casts in urine of, 380
contracted, casts in urine in, 374
edema with, 399
gallop-rhythm in, 191
hard pulse in, 210
hyaline casts in urine of, 380
polyuria in, 366
retinitis albuminurica in, 547
-disease, dropsy of, 398
engorgement of, casts in urine of, 380, 381
lateritious sediment in urine, 387
Kidney, exclusion of one, polyuria from, 36
fatty. See *Fatty kidney.*
floating, 358
horseshoe, 356
injuries to, hematuria from, 375
large white, adipose elements with renal casts in urine of, 381
edema with, 399
fat in urine of, 378
renal epithelia in urine of, 379
movable, 358
percussion of, 358
tumors of, detection of, 357
wandering, 357
Kidneys, anatomy of, 355
engorgement of, albuminuria in, 394
examination of, 355
inspection of, 356, 357
palpation of, 356, 357
percussion of, 356
tenderness on palpation, 357
Kissing as a source of bacterial infection, 257
Kistermann on bismuth-test for sugar, 402
Kitasato, direct culture of tubercle bacilli, 162
Klemperer on course of separation of albumins in urine, 396
Klemperer's oil method for determining rapidity of evacuation of stomach-contents into duodenum, 322
Knee-phenomenon, 459
Knife-blade crystals in urine, 389
Knoll's rubber bottle, 141
Knowledge of form, testing of, 446
Koch's tuberculin, effect of, on sputum of tuberculosis, 162
use of, in diagnosis, 164
Kosselt on peptone in sputum, 164
Krehl on heart-sounds, 184
on "recoil" of pulse-curve, 212
Külz on casts in urine of diabetic coma, 380
on oxybutyric acid in urine of diabetes, 407
Kussmaul on ataxic aphasia, 506
on congenital displacement of pylorus, 271
on effect of respiration on jugular veins in mediastino-pericarditis, 226
on the tract for speech-motions, 505
"peristaltic unrest" of stomach, 272
word-deafness, 506
Kussmaul's test-meal, 312
Kuttner on use of illuminating sound to introduce water into stomach, 277
Kyphoscoliosis, 78
a cause of hypertrophy of right ventricle, 170
dyspnea in, 85
spleen can be felt in, 298
Kyphosis, 78
acute-angled, effects of, 434
from paralysis of erector trunci, 495

LAACHE on leucin and tyrosin in urine of pernicious anemia, 390
Lachrymal nerve illustrated, 448
Lactalbumin in digestion, 306
Lactic-acid fermentation in digestion, 305
in stomach, determination of, 318

Lactose in urine, 406
Laënnec, developed auscultation, 118
reference to, as user of percussion-hammer, 93
Laënnec's pectoriloquy, 135
"Lagging," detection of, by palpation, 91
Lagophthalmus, 493
Lalopathy, 502
Landerer on cause of edema, 48
on constant circumscribed tenderness in gastric region, 273
Laryngeal catarrh, signs of, 538
crises, 525
fremitus, 67
mirror, 535
muscles, functions of, 494
paralysis of, 494
Laryngitis as a symptom of disease, 68
hypoglottica, 538
paralysis of thyreo-arytænoideus internus in, 542
paralysis of arytænoideus transversus in, 541
phlegmonous, 539
swelling of larynx in, 538
Laryngoscopic image or view, 536
Laryngoscopy, 535
instruments for, 535
sources of light in, 535
Larynx, abnormal redness of mucous membrane of, 538
carcinoma of, 541
casts of, 149
examination of, 67
fibroma of, 541
gummata of, 540
laryngoscopic examination of, 535
motor center for, 416
movements of, 537
new formations of, 541
normal color of, 537
percussion-sound over, 105
paleness of mucous membrane of, 537
papilloma of, 541
paralysis of, 68
of muscles of, 541
pathological conditions of, 537
relation of disease of, to nervous disease, 525
sarcoma of, 541
scars of, 540
spasm of muscles of, 541
stenosis of, 67
symptoms of disease of, 68
swelling of, when occurs, 538
syphilitic infiltration of, 540
ulceration of, 539
tubercular ulceration of, 540
tympanitic sound over, 97
ulceration of, 539
cough in, 143
Lateritious sediment of urine, 363
Latissimus dorsi, paralysis of, testing of, 498
Laveran's bodies in malarial blood, 247
Law of contraction, 478
Lead colic, hard pulse in, 210
pulsus tardus in, 210
Lead colic, scaphoid abdomen in, 279
in urine, 411
-paralysis, effect of, 498
reaction of degeneration in, 484
-poisoning, black vomit in, 328
chronic, teeth and gums in, 253
headache in, 447
neuralgia in, 447
Leanness, significance of, 31
Leg, motor center for, 417
paralysis of muscles of, 500, 501
Lenticular nucleus, blood-supply of, 422
disease of, in chorea, 492
Leo on evacuation of milk from stomach of infants, 357
on prevention of fermentation in stomachs of nurslings, 307
on stomach-digestion of nurslings, 308
Lepra bacillus, 552
Leptothrix buccalis in saliva, 257
in sputum, 150, 158
in urine, 385
in pharynx, 263
Lethargic stage of hypnosis, over-excitability of nerves and muscles in, 486
Letzerich and Berger, microbe of whooping-cough, 164
Leube's method of obtaining gastric secretion, 323
test-meal, 312
Leucin in urine, 390
Leuckart on tænia cucumerina in children, 343
Leukemia, albuminuria in, 394
ascending venous pulse in, 229
diminution of red corpuscles in, 238, 244
enlargement of spleen in, 298
hematuria in, 376
hemorrhage from fundus of eye in, 547
leukocytes in, 242
lienal-myelogenous, leukocytes in blood of, 242
lymphatic, blood in, 244
myelogenous, 242
paleness in, 36, 37
retinal, changes in, 547
surface of spleen in, 299
tumors of spleen in, 298
uric acid increased in, 392
Leukocytosis, 242
cachectic, 242
hydremic, 242
inflammatory, 242
physiological 242
Levulose in urine, 406
Levator anguli scapulæ, paralysis of, effect of, 497
Levy on effect of presence of Fränkel's pneumococcus in pleural exudates, 138
Lewis and Miller on bacillus resembling comma bacillus in tooth-mucus, 351
Leyden, hemisystolia, 175
on double positive venous pulse in hemisystole, 229
on fatty acids in sputum of gangrene of lungs, 164

Leyden on tyrosin in sputum, 157
Lichtheim on edema, 47
on mental condition in atactic aphasia, 507
Lichtheim's diagram of aphasia, 511
Lieben on test for acetone in urine, 409
Liebermeister on pulse in fever, 205
on testing the accuracy of a thermometer, 51
Lientery stools, 337
Life, manner of, in the causation of disease, 21
Light reflex of pupil, 518
rigidity of pupil, 518
Lime-salts in feces, 349
Linea costo-articularis, 296
Lipaciduria, 407
Lipemia, 245
Lipomatosis cordis, weak apex-beat in, 174
Lips, examination of, 253
Lipuria, 372, 406
Literal agraphia, 513
aphasia, 507
Litten on casts of micrococci in urine, 385
on diaphragmatic phenomenon, 74
Liver, abscess of, tenderness in, 290
acute yellow atrophy of, cutaneous hemorrhage in, 46
affections of urobilinuria in, 369
anatomy of, 286
biliary engorgement of, tenderness in, 290
cancer-navel upon, in carcinoma, 292
carcinoma of, tenderness in, 290
cirrhosis of, tenderness in, 290
consistence of, in various diseases, 292
constricted, palpation of, 290
downward displacement of, 288, 289, 291
dulness, 292, 292
displacement of boundaries of, 294
relative, 108
engorged, increased consistence of, 292
engorgement of surface of, 291
of, tenderness in, 290
enlargement of, causes and signs of, 289
dulness in, 295
expansion of thorax in, 77
from general venous engorgement, 224
liver-pulse in, 289
projection of right hypochondrium in, 288
smallness of Traube's space in, 275
examination of, 286–296
exploratory puncture of, 292
form of, 291
hypertrophic, surface of, 291
inspection of, 288
-kidney angle, 355
lobulated, 291
mobility of, boundaries of, 294
palpation of, 289
pathological relations of boundaries of, 294
percussion-limits of, 293, 294
percussion of, 292
pulse, 221
arterial, 228
in enlargment of liver, 289
palpation of, 228
venous, 228
size and form of, 290
Liver, square position of, 295
surface of, in various diseases, 291
syphilitic, tenderness of, 290
tenderness of, 290
tumors of, inspection of, 289
upward displacement of, 289, 295
dulness in, 295
wandering, 289
absence of liver-dulness in, 296
"Livid skin," 38
Lobes of the lungs, boundaries of, 72
Lobulated liver, 291
Local asphyxia in nervous diseases, 526
disease, diagnosis of location of, 532
sense, testing of, 440
Localization, power of, testing of, 440
Löffler's bacillus, determination of, 260–262
diphtheria bacillus in sputum, 164
methylene-blue, 262
Logwood, color of urine, after taking, 371
Lordosis, 434
Louis, angle of, 73
Löwenfeld on contractions in reaction of degeneration, 483
"Luftschlucken," 310
Lugol's solution, 322
Lumbar cord, diseases of, residual urine in, 368
enlargement of cord, location of, 434
puncture, 434
Lumbo-inguinal nerve illustrated, 451
Lung-cavities, bronchial breathing over, 126
metallic heart-sound in, 191
metallic sound over, 98
plegaphonia over, 135
tympanitic sound over, 97, 113
-dyspnea, 85
-fistula, sound of, 131
-heart boundary, 106
infiltrated, plegaphonic sound in, 135
-kidney boundary, 106
-liver boundary, 106
sequestra in sputum, 147
-sound, 95
-spleen boundary, 106
thickening of, ringing râles in, 130
-tissue, decreased tension of, abnormally loud percussion-sound in, 116
in sputum, 147
retracted, cracked-pot sound in, 116
thickening of, bronchial breathing in, 125
Lungs, abnormal percussion-sounds over, 109
abnormal position of border of, 109
abscess of, shrinking of chest-wall in, 78
anatomical boundaries of, with reference to the thorax, 69
auscultation of, 118
compression of, bronchial breathing in, 125
contraction of, a cause of hypertrophy of right ventricle, 170
drawing-in of side of thorax in, 77
deadened resonance over, 109
diminished volume of, recognized by lung boundaries, 118
diminution of motility of, 118
diseases of, dyspnea in, 85
dulness over, 109

Lungs, edema of dyspnea in, 85
examination of, 68
gangrene of, shrunken chest-wall in, 78
hemorrhage of, 146
infarctions of, percussion resonance in, 110
infiltration of, resonance in, 110
non-tympanitic sound over, 99
normal boundaries of, 105
percussion boundaries of, 106
sound of, 105
percussion of, 103
shrinking of, a cause of displacement of apex-beat, 173
thickening of, resistance over, 102
Lupus a cause of catarrh of larynx, 538
Lustgarten on bacteria in normal urine, 361
Lymphatic glands of neck, examination of, 260
Lymphocytes, 242
Lymphosarcoma of spinal cord, hemialbumose in urine of, 398
Lysis defined, 61
Lyssa, increase of tendon-reflexes in, 461

MACROCEPHALUS, 430
Macrocytes, 240
nucleus-containing, 240
Main en griffe, 499
Malachowski's method of staining malarial parasites in blood, 248
Malaria, enlargement of spleen in, 298
fever in, 57
hemoglobinuria in, 370
intermittent fever of, 63
melanemia after, 245
neuralgia in, 447
Malarial cachexia, paleness in, 36
parasites in blood, 247
Malignant edema, bacilli of, 550
Mal perforant, 530
Mammillary lines, 69
Mancgegang, 491
Mannaberg on Malachowski's staining malarial parasites in blood, 248
on bacteria in normal urine, 361
Marasmus defined, 31
Marey's pneumograph, 141
Marie on acromegalia, 531
Martins' method of showing relation of apex-beat to heart-cycle, 172
Mast-cells, 242
staining of, 245
Mastication, muscles of, function of, 493
paralysis of, 494
Masturbation, spermatorrhea from, 414
Matterstock, sound over crural artery in lead-poisoning, 222
Maximal thermometers, 51
Measles, acute laryngitis in, 68
acute nasal catarrh in, 66
cutaneous hemorrhage in, 46
entrance of, through tonsils, 258
epileptiform convulsions in, 490
recurrence of, 22
Measurement, methods of, 140
Meat-poisoning, bacillus enteritidis in, 554
Mechanical excitability of muscles and nerves, 485
Mediastinal emphysema, crepitation of, 200
pericarditis, systolic drawing in of intercostal spaces in, 177
tumors a cause of cyanosis, 38
of displacement of apex-beat, 173
venous engorgment from, 224
Mediastinitis, fibroid, diastolic collapse of cervical veins in, 229
effect upon venous circulation, 226
Mediastinopericarditis, effect of respiratory motions on jugular veins in, 226
Medicines, discoloration of urine from, 371
which cause polyuria, 367
Medulla oblongata, disease of, salivation in, 256
softening of, recurrent paralysis in, 543
Melæna neonatorum, bloody vomit in, 329
Melancholia attonita, catalepsy in, 492
Melanemia, 245
Melanosis, arsenical, 43
Melas-icterus, 40
Mellituria, 367
Memory, testing of, 436
Ménière's disease, tinnitus aurium with dizziness in, 523
Meningitis, albumin in cerebrospinal fluid in, 435
anosmia in, 524
breathing in, 81
catalepsy rare in, 492
central vomiting in, 326
cerebrospinal, temperature with, 64
Cheyne-Stokes' respiration in, 82
choked disk in, 547
disturbance of consciousness in, 437
fibrin in cerebrospinal fluid in, 435
from disease of ethmoid bone, 525
hard pulse in, 210
headache in, 446
herpes with, 45
irregular fever in, 64
metastatic, Fränkel's pneumococcus in, 163
neuroretinitis in, 547
opisthotonos in, 29
pain in spine in, 447
posture in, 29
pulse in, 210
purulent, pus in cerebrospinal fluid in, 435
rigidity of neck in, 434
scaphoid abdomen in, 279
sensibility of cranium to pressure in, 433
sensitiveness of spine to pressure in, 434
spinal, rigidity of spinal column in, 474
herpes zoster in, 529
tuberculous, bacilli in cerebrospinal fluid in, 435
Mental disease, hemidrosis in, 34
exertion, glycosuria in, 402
Mercurial poisoning, salivation of, 256
Mercury in urine, 411
poisoning, glycosuria in, 401
headache in, 447
looseness of teeth in, 253
neuralgia from, 447

Mesocardia, 182
Mesogastrium defined, 268
illustrated, 268
Metallic after-sounds, 98
heart-sounds, 191
murmurs, 196
pericardial splashing, 200
râles, 128
sound, 98
Metamorphosing breathing, 127
Metapneumonic exudations, micro-organisms in, 138
Meteorism, absence of liver-dulness in, 296
expansion of thorax in, 77
metallic heart-sounds in, 191
percussion-sound in, 280
Meteorismus peritonei, 281
recognition by percussion, 284
Methemoglobin in blood in poisoning with chlorate of potash, 236
in urine, 400
Methyl-violet test for hydrochloric acid in stomach, 317
Microbe of whooping-cough, 164
Microcephalus, 431
Micrococci, casts of, in urine, 385
Micrococcus gonorrhœæ, 549
staining of, 549
tetragenus, 159
ureæ causes alkaline fermentation of urine, 383
liquefaciens causes alkaline fermentation of urine, 383
Microcytes, 240
Microcythemia, 240
Micro-organisms in blood, staining of, 247
in feces, 349
in mouth, 257
in pleural exudations, examination for, 137
in urine, 361
Microscopic examination of blood, 237
of feces, 347
of mouth-contents, 256
of sputum, 150
Micturition, difficult, a cause of diminished quantity of urine, 368
Miescher's improved blood-mixer, 244
Mietz on determination of free hydrochloric acid in stomach, 319
Migraine, 447
from tinnitus aurium, 523
hemidrosis in, 34
hyperacidity in, 325
inequality of pupils in, 517
polyuria in, 367
subjective sensation of light in, 522
unilateral paleness or redness of head in, 526
vomiting with, 527
Migrène ophthalmique, 522
Miliaria, 45
Milk-curdling ferment, secretion of, 306
particles of, in sputum, 150
Mind, condition of, mode of examining, 436
Minimal contraction, 473, 475
Minkowski on oxybutyric acid as a cause of diabetic coma, 407
Minkowski's researches on formation of bile-pigment in geese and ducks, 40
Miserere, 330
Mitral insufficiency. See *Insufficiency.*
disease, heart-disease cells in sputum of, 152
venous engorgement in, 224
stenosis. See *Stenosis.*
valve, auscultation of sound of, 184, 185
Moist cough, 143
râles, 129
Monas in sputum, 158
Monocrotic pulse, 213
Monoplegia accompanying Jackson's epilepsy, 491
defined, 452
from lesion of corona radiata, 425
Moore's test for sugar in urine, 403
Morbus Werlhofii, hematuria in, 376
Morenheim's depression, 68
Moritz on action of stomach, 307
on constituents of urinary crystals, 386
on the error of Reuss's method of estimating albumin, 140
on fermentation-test for sugar, 404
on free hydrochloric acid in digestion, 306
on glycosuria, 402
on interrupted Wintrick's change of sound, 114
on sugar in urine in health, 366
Morphia, effect of, on pupil, 517
-poisoning, glycosuria in, 401
Morphin in urine, 411
-poisoning, Cheyne-Stokes' respiration in, 83
Motility, disturbances of, 452
Mosso on Cheyne-Stokes' respiration in healthy sleep, 83
Motor activity of stomach, diminution of, 309
aphasia, 507
points, 469
of head and neck, 469
on arm, 470, 471
on leg, 474
on thigh, 472
upon back of leg, 473
speech-center, location of, 505
tracts and centers, 416
tract, central, 416
peripheral, 418
Mould in sputum, 164
Mouth-contents, bacteria in, 257
microscopic examination of, 256
examination of, 252
floor of, examination of, 255
mucous membrane of, examination of, 255
odor from, importance of, 252
tympanitic sound over, 97
Movable kidney, 358
Mucin in urine, 399
test for, 393
-like substance in urine, 393
Mucopurulent sputum, 145
Mucous corpuscles in sputum, 150
in vomit, 331
membrane of intestine, disease of, poor resorption of food in, 335

Mucous membrane of intestine, shreds of, in feces, 340
of mouth, examination of, 255
reflexes of, 458
sputum, 144
stools, 337
vomit, 328
Mucus in stools, 348
in urine, 375
chemical proof of, 375
Muguet in sputum, 150
Mulberry calculi, 391
tongue, 254
Müller (F.) on urobilin icterus, 42
on eosinophilous asthma-cells, 157
on demonstration of ascitic fluid, 284
on effect of carcinoma on secretion of hydrochloric acid, 325
on formation of urobilin in intestines, 43
on imperfect absorption of fat in enteritis, 354
on quantity of fluid in bladder needed to give dulness on percussion, 360
on resorption of food in enteritis, 335
Multiple neuritis. See *Neuritis.*
sclerosis. See *Sclerosis.*
Murexid test for uric acid, 387
Muriatic acid. See *Hydrochloric acid.*
Murmur, double, over arteries, 222
of insufficiency, 192
subclavian, 222
venous, 230
Murmurs, anemic, 197
combination of several, 196
diastolic and systolic, 193, 196, 197
differential diagnosis between pericardial and endocardial, 199
differentiation of, 196
of inorganic from organic, 198
endocardial, 191
inorganic, 197
loudness of, 195
metallic, 196
pericardial, 198
that may be felt, 196
Musca vomitoria, larva of, in urine, 383
Muscles, active spasm of, 456
auxiliary, of inspiration and expiration, 86
diminution of volume of a symptom of degeneration of motor tract, 425
electrical examination of, 462
electric stimulation of, 468
mechanical excitability of, 485
motor points of, 469
of face, function of, 492
of larynx, paralysis of, 541
of mastication, paralysis of, 494
of throat and neck, functions of, 495
paralysis of, 495
of tongue, functions of, 494
of upper extremity, 496
rigidity of, 456
supplied by radial nerve, elcctrical examination of, 475
voluntary, innervation, function, and diseases that disturb them, 492
Muscular dystrophia, 455
sense, testing of, 444
Musculospiral nerve, paralysis of, 451
Musical notes, conception of, 504
Muttering delirium, 438
Mydriasis, 517
Myelitis, allochiria in, 443
decreased tendon-reflexes in, 461
transversa, clonic spasms in, 490
cystitis in, 528
extreme atrophy of legs in, 454
increased tendon-reflexes in, 461
involuntary discharge of urine in, 528
Myocarditis, arhythm in, 207
fibroid, retardation of pulse in, 204
heart-disease cells in sputum of, 152
pulse in, 217
weakened apex-beat in, 174
Myosis, 517
Myositis ossificans, cyanosis in, 39
Myotonia congenita, 483
increased mechanical excitability in, 485
tonic spasms in, 490
Myotonic reaction, 483

NAILS, anomalies of, in peripheral paralysis, 530
Naphthalin, color of urine after taking, 371
in urine, 411
Nasal mucous membrane, disease of, anosmia from, 524
polypi, relation of, to nervous disease, 525
septum, appearance of, 543, 544
sound, use of, 544
specula, varieties of, 543
Naunyn, after-sensibility, 443
and Minkowski, researches on formation of biliary pigment in ducks, 40
on fever from injury of cervical spinal cord, 525
on murmur of mitral insufficiency, 192
Negative venous pulse, 226
Neisser on diagnosis of gonorrhea, 384
Nephritis a cause of hypertrophy of left ventricle, 170
acute, bacteria in urine in, 385
albuminuria in, 394
anuria in, 368
character of pus-sediment in, 378
chlorid of sodium in urine diminished in, 392
cyclic albuminuria in, 396
cylindroids in urine of, 375
decrease of hydrochloric acid in stomach in, 324
diminished urea in urine of, 392
diminution of phosphate in urine of, 392
edema in, 47, 48
epithelial casts in urine of, 382
erysipelatous cocci in urine of, 385
granular casts in urine of, 382
hematuria in, 375
infectious, entrance of, through tonsils, 258
low specific gravity of urine in, 372
mucin in urine of, 399
nucleo-albumin in urine with, 399
odor of vomit in, 332
pulse-tracing in, 212
renal epithelia in urine of, 379

Nephritis, retardation of pulse in, 203
retinitis albuminurica in, 547
sequela of scarlet fever, 22
sweat in, 34
tenderness over kidney in, 357
turbid urine in, 371
urea in saliva of, 256
white blood-corpuscles and pus in urine of, 377
Nephrolithiasis, brick-dust deposit in, 387
hematuria in, 376
Nerve, faradic examination of, 473
Nerves, electrical examination of, 462
electric stimulation of, 467
galvanic examination of, 475
mechanical excitability of, 485
of head, distribution of, 448
of lower extremities, distribution of, 451
of shoulder, arm, and hand, 449
peripheral, and their surroundings, 436
sensory cutaneous, distribution of, 448
Nervous diseases, diagnostic value of symptoms in, 531
differential diagnosis between functional and anatomical, 533
examination of seat of, 430
polyuria, 367
system, anatomy of, 416
examination of, 414
Nervousness from intestinal parasites, 527
hypersecretion of gastric juice in, 325
palpitation and pain of heart in, 526
sensibility of cranium to pressure in, 433
subjective sensibility of hearing in, 523
syncope in, 438
Neubauer on specific gravity of urine as a guide to amount of solid constituents, 372
Neukirch, explanation of Williams's tracheal tone, 106
Neuralgia, 447
bradycardia in, 204
glycosuria from, 402
in diabetes mellitus, 528
intercostal, 90
tenderness of nerve in, 448
Neurasthenia, cerebral, vertigo in, 438
facial phenomenon in, 486
headache of, 447
pain in spine in, 447
phosphaturia in, 389
polyuria in, 367
retardation of pulse in, 204
spermatorrhea from, 414
spinalis, disturbance of genital function in, 529
Neuritis, acute degenerative, disunion of atrophy and paralysis in, 456
ataxia in, 488
in diabetes mellitus, 528
multiple, bladder disturbances absent in, 528
diminished irritability in, 485
neuralgia in, 447
optica, 513
peripheral, a cause of cyanosis, 39
degenerative atrophy in, 454
Neuritis, peripheral, herpes zoster in, 529
quickened pulse in, 526
reaction of degeneration in, 479, 484
sensitiveness of peripheral nerves in, 448
significance of, 546
thickening of nerve from, 436
Neuro-fibroma, condition of nerve with, 436
Neuroma, condition of nerve in, 436
Neuro-retinitis albuminurica, 547
diabetic, 547
when occurs, 547
Neuroses, frequent pulse in, 207
increase of tendon reflexes in, 461
local sweating in, 34
Neutrophile cells, staining of, 245
Nicotin-poisoning, headache in, 447
neuralgia in, 447
palpitation and pain of heart in, 526
strengthened heart-beat in, 174
vertigo in, 438
Niemeyer's solid stethoscope, 120
Night-sweats of phthisis, 34
Nitrobenzol-poisoning, color of blood in, 233
odor of vomit in, 332
Nitrozo-indol reaction, value of, 554
Nocturnal enuresis, 528
Noma, description of, 255
Non-tympanitic percussion-sound, 95
sound, intensity of, 99
where heard, 99
Nose, affections of, relation to nervous disease, 525
anomalies of, 66
-bleed, 66
disease of, migraine in, 447
examination of, 66
suppuration of, as a cause of meningitis, 433
Nothnagel on mucus in stools, 337, 338
on staining of micro-organism of feces, 349
Nothnagel's thermesthesiometer, 441
Nourishment, food-value of, determination of, 353
Nubecula of urine, 363
Nuclear paralyses, 419
Nucleo-albumin in urine, 399
Nutrition as affected by nervous disease, 525
disturbances of, 453
examination of, 30
Nutritional tone of muscles, disturbance of, 453
Nylander's test for sugar in urine, 402
Nymphomania, 439
Nystagmus, 516

Obermayer's test for indican in urine, 370
Obernier on rise of temperature in a person running, 53
Obersteiner, allochiria, 443
Obliquus inferior, paralysis of, effect of, 516
superior, paralysis of, effect of, 516
Oblongata, diseases of, glycosuria in, 402
effect of lesion of, 425
Obstipation, 333, 334
indicanuria from, 369
large stools after prolonged, 335

Obstruction of Eustachian tube, drawing-in of ear-drum from, 546
Occlusion of bowels, fecal vomiting, 330
of intestine, indicanuria in, 369
Occupation as a factor in causation of disease, 21
Oculomotorius, course of, 418
total paralysis of, effect of, 516
Odor from the mouth, 252
of expectoration, 147
of sputum, 147
of stools, 336
of urine, pathological, 373
Oidium albicans, coating of tongue by, 255
Old age, nutrition of skin in, 32
retardation of pulse in, 204
Oligocythemia, 238
Oliguria in hysteria, 528
Omentum, carcinomatous knots in, 303
examination of, 302
shrinking of, in tuberculosis and chronic peritonitis, 302
Onanism, concealed, 20
Ophthalmia neuroparalytica, 448
Ophthalmoplegia externa, 516
Ophthalmoscopy, method of performing, 546
Opisthotonos, 434
a form of tonic spasm, 490
how produced, 496
in meningitis, 29
Optic nerve, atrophy of, mydriasis with, 517
pressure on, choked disk from, 547
primary atrophy of, 547
thalamus, effect of diseases of, 427
tract, 420
lesion of, hemianopsia in, 521
Organic heart-murmurs, 191
Orthopnea, 29
effects of, 29
in phthisis, 29
Osmic paresthesia, 524
Osteomyelitis, staphylococcus pyogenes in, 548
Otitis, Fränkel's pneumococcus in, 163
Otoscopy, method of performing, 545
Ovarian hyperesthesia, 529
Ovary, painful, 529
Oxalate of lime in sputum, 157
in urine, 387
Oxaluria, 389
Oxybutyric acid a cause of diabetic coma, 407, 408
in urine, 407
testing for, 408
Oxygen, deficiency of, a cause of cyanosis, 38
in blood, diagnostic importance of, 233
Oxyhemoglobin, absorption-band of, 235
effect of, on color of blood, 233
Oxyuris vermicularis, 344
in urine, 383

PAIN a cause of rapid breathing, 84
after use of sound in esophagus, 265
diminution of power of motion from, 452
electric sensibility to, 441
gradual increase of sensibility to, 443
in ear from diseased teeth, 523
Pain in iliac fossa, left, 278
right, 277
in peritonitis, 282
in spine, 447
mydriasis in, 517
on palpation of spleen, 298
on pressure over intestines, 277
on swallowing in stenosis of esophagus, 264
sensibility to, testing of, 441
spontaneous, 446
syncope from, 438
thoracic, caused by pressure, 89
with the stools, 335
Painful ovary, 529
Palate, examination of, 257, 258
microscopical and bacteriological, 260
lodgement of bacteria in, 257
soft, innervation of, 494
Pale skin, 35
Paleness, 35
Palpation of arteries, 220
of liver, 289
of liver-pulse, 228
of mouth, 252
of pulse, 201
of stomach, 270
of thorax, 89
of veins, 223
of vocal fremitus, 133
Palpitation, nervous, strengthened heart-beat in, 174
Pancreas, examination of, 302
extirpation of, a cause of diminished fat-digestion, 348
tumor of, 302
Pancreatic juice, deficiency of, absorption of fats in, 335
Panizza on origin of epithelium in sputum, 151
Papilloma of larynx, 541
Para-anesthesia, 443
Paradoxical contractions, 486
Paragraphia, 513
Paralysis, 452
agitans, sensations of heat in, 526
tremor of, 489
atonic, 451
atrophic, 457
degenerative, 419
atrophic, 453
fibrillary contractions in, 490
partial reaction of degeneration in, 484
bulbar, reaction of degeneration in, 484
central, arrest of growth of bones after, 530
cerebral facial, 493
degenerative atrophic, indicated by reaction of degeneration, 483
disseminated, reaction of degeneration in, 479
dissociated, from lesion of corona radiata, 425
disturbance of conception of motion in, 446
examination for, 452
extent of, 452
facial, 493
frequent pulse in, 207

Paralysis from affections of cortex of brain, 436
increased irritability in, 485
intermitting general, 485
nuclear, 419
of abdominal muscles, effects of, 495
of accommodation, 515
of arytænoideus transversus, 541
of bladder, 528
of crico-arytænoidei postici, 542
of crico-thyroidei muscles, 543
of diaphragm, 496
a cause of cyanosis, 39
of erector trunci, 495
of extensor muscles of hand, 498
of facial nerve, symptoms of, 493
of hand and fingers, characteristic positions in, 499
of heart, pulse in, 207
of muscles of arm, 498
of eye, 513, 514
significance of, 516
of larynx, 541
of leg, 500
of lower extremity, 500
of mastication, 494
of shoulder, 496
of speech, 502
of trunk and scapula, 497
of oculomotorius, effect of, 515
of pharyngeal muscles, 494
of phrenic nerve, shown by palpation, 91
of recurrent nerves, 542
of respiratory muscles, dyspnea from, 85, 525
of soft palate, 494
of stapedius, disturbance of hearing in, 523
of tensor of vocal cords, 543
of thyreo-arytænoideus internus, 541
of tongue, 494
phenomena of, 452
progressive, imbecility in, 439
mydriasis with, 517
reaction of degeneration in, 484
relaxed, as a sign of lesion of cortical center, 425
spastic, 419
spinal, dorsal flexion of foot in, 492
unilateral hypoglossal, 494
vasomotor disturbances in, 526
Paralytic dementia, disturbance of hand-writing in, 513
thorax, 76
Paralyzed muscles, tonus of, 456
Parametritic abscess, pus in urine from, 377
Parametritis, position in, 30
Paraphasia, 507
Paraplegia, inferior, 453
Parasites, animal, in sputum, 157
in urine, 382
of alimentary canal, 340
intestinal, as a cause of nervous disease, 527
vegetable, in feces, 349
in urine, 383
Parasternal lines, 69
Parcsis, defined, 452
recognition of, 452
Paresthesia, 446
osmic, 524
Parietal boundaries of organs, determining of, 102
cavities, cracked-pot sound in, 116
Parosmia, 524
Parotid gland, inflammation and swelling of, 256
Paroxysmal tachycardia, 207
Partial epilepsy, 490
Patellar reflex, 459
Pathogenic fungi in stools, 349
Patient, general examination of, 28
position, attitude, and posture of, 28
previous history of, 20
psychical condition of, 28
Patients, examination of, 24
Pectoralis major, paralysis of, testing of, 498
Pectoriloquy, Laënnec's, 135
Peliosis rheumatica, cutaneous hemorrhages in, 46
Pemphigus, nervous origin of, 529
Pendulum-rhythm, 190
Penis, irritation of, effects of, 529
Penzoldt on crepitation in expiration, 132
on picric-acid test for albumin in urine, 395
Penzoldt's method for examining absorption in stomach, 323
modification of Gmelin-Rosenbach test, 401
Pepsin, diminished secretion of, effect on digestion, 308
secretion of, by stomach, 305
Peptone, absorption of, by stomach, 307
in sputum, 164
in urine, 398
test for, 322
Peptonuria, 398
Perception of movement, testing of, 444
Percussion, comparative, 104, 105
difference between weak and strong, 95
direct, 92
effect of feeble, 100
finger-, 93
general remarks upon, 92
-hammer, 93
history and methods of, 92
immediate, 92
indirect, 92
mediate, 92
of abdomen in disease of peritoneum, 283, 284
of esophagus, 267
of heart, 177
methods of, 177, 180
of intestine, 280
deep, 281
of liver, 292
of stomach, 273
of thorax, 92
-sound, conditions that determine quality of, 96
qualities of, 94
variation of, in individuals, 105
three methods of, 93
to determine parietal boundaries of organs, 102
to determine resistance of heart, 180

Percussion, topographical, 102
Perforating disease of the foot, 530
Pericardial adhesions, absence of apex-beat in, 175
exudation, cause of weakening of heart-sound, 189
cyanosis from, 38, 39
friction-sounds, 198
murmurs, 198
distinguished from endocardial, 199
splashing, 200
Pericarditis adhesiva, divided second apex-sound in, 190
heart-disease cells in sputum of, 152
pulsus paradoxus in, 209
systolic drawing-in of intercostal spaces in, 177
downward displacement of liver, 289
exudativa, abnormally loud percussion-sound in, 116
area of heart-dulness in, 181
bulging in neighborhood of heart in, 176
disappearance of apex-beat in, 174
effect of, on position of liver, 296
pulse in, 217
tympanitic resonance in, 112
friction-sound in, 199
mediastinal, systolic drawing-in of intercostal spaces in, 177
pulse in, 218
venous engorgement in, 224
Pericardium, diseases of, paleness in, 36
distention of, expansion of thorax in, 77
dryness of, friction-sounds in, 199
fluid in, effect on heart-dulness, 181
tuberculosis of, friction-sound in, 199
Perichondritis arytænoidea, 540
laryngea, 540
pus in larynx from, 539
Pericystic abscess, pus in urine from, 377
Perinephritic abscess, detection of, 357
pus in urine in, 377
Perinephritis, palpation of kidney in, 358
purulent, tumor of kidney in, 357
tenderness with, 357
Perineuritis, condition of nerve in, 436
Periosteal reflexes, 460
Periostitis, neuralgia due to, 447
of ribs, pain with, 90
Peripheral irritation, sensitiveness to, 439
motor tract, 418
nerves, examination of, 435
tendon reflexes decreased in disease of, 461
neuritis, herpes zoster in, 529
local asphyxia in, 526
paralysis, 419
delayed sensibility in, 443
sight, testing of, 520
Peripleuritis, deadening of chest-resonance in, 112
Periproctic abscess, purulent stools from, 339
Periproctitis, pain at stools with, 335
Perisplenitis, enlargement of spleen in, 298
Peristalsis, intestinal, visible, 279
Peristaltic motions in stomach, importance of, 272
Peristaltic unrest of stomach, 272
Peritoneal cavity, air in, 285
exudate, anuria during formation of, 367
fluid, encysted, 283, 284
examination of, 285
friction-sound, 285
Peritoneum, air in, absence of liver-dulness in, 296
bands in, after tapping abdomen, 285
diseases of, inspection of abdomen in, 281
examination of, 281
palpation of, through vagina, 283
Peritonitis, ascites that moves about in, 282
cause of cyanosis, 39
Cheyne-Stokes respiration in, 83
chronic, constipation alternating with diarrhea in, 334
diagnostic points of, 282
shrinking of omentum in, 302
tenderness of liver in, 290
distention of abdomen in, 278
expansion of thorax in, 77
fecal vomiting, 330
Fränkel's pneumococcus in, 163
from paralysis of intestine, obstipation in, 334
hardness of abdomen in, 282
indicanuria in, 369
leukocytosis in, 242
local, the detection of, by palpation, 91
pain in, 282
pain on palpation of intestines in, 277
pulsus tardus in, 210
purulent, staphylococcus pyogenes in, 548
subphrenic, defined, 284
friction-sounds in, 200
surface of liver in, 291
tubercular, enlargement of spleen in, 298
tympanitic sound in, 112
vomiting in, 327
Perityphlitic abscess, pus in urine of, 377
Perityphlitis, position in, 30
Pernicious anemia. See *Anemia.*
hemorrhage in fundus of eye in, 548
Peroneus nerve, electrical stimulation of, 472
-paralysis, 501
Perspiration, 32
in illness, 33
insensible, 33
local, 34
morbid alterations of, 33
of feet, loss of, relation to nervous diseases, 529
office of, 33
qualitative alterations of, 34
relation of, to nervous disease, 529
varieties of, 33
Perturbation, critical, 61
Petechiæ, 44, 45
Pettenkofer's test for bile-acids in urine, 401
Petters on odors of acetone in urine, 373
Pfeiffer (R), influenza bacillus, 552
Pflüger's law, 467
Pharyngeal muscles, paralysis of, 494
Pharyngomycosis leptothricia, 263
Pharyngoscopy, 544
Pharyngosis leptothricia, 263

Pharynx, anesthesia of, 527
examination of, 260
microscopical and bacteriological, 260
growth of leptothrix in, 263
Phase de grand mouvements, 491
Phenyl-hydracin test for sugar in urine, 403
Phlegmon of larynx, appearance of, 538
Phloroglucin-vanillin reaction, 317
Phonation, vibrations of glottis in, 133
Phonendoscope, value of, 120
Phosphate, deposit of, in urine, 363
of lime in urine, 389
Phosphatic calculi, 391
Phosphaturia, 389
Phosphorus-poisoning, cutaneous hemorrhage in, 46
leucin and tyrosin in urine of, 390
odor from mouth in, 253
odor of vomit in, 332
peptonuria with, 398
Phrenic nerve, unilateral paralysis of, detected by palpation, 91
Phthisical thorax, 76
Phthisis, accommodation in, 85
aspergillus fumigatus in sputum of, 150
asymmetrical breathing in, 81
diminished inspiratory pressure in, 141
diminution of size of apex of lung in, 118
ferment in sputum of, 164
form of thorax in, 76
friction-sounds in, 133
lagging in, 91
neuralgia in, 447
night-sweats of, 34
odor of sputum in, 147
orthopnea in, 29
sudden stopping of cough in, 142
systolic subclavian murmur in, 222
tenderness with, 90
thoracic tenderness in, 90
vital capacity of lungs in, 141
Picric acid, color of urine after taking, 371
test for albumin in urine, 395
Pigeon-chest, 78
Pill-maker hand, 489
Pilocarpin, effect of, on pupil, 517
Pio Foà's coccus in sputum, 162
Piorry, reference to, as a reviver of percussion, 92
Plasmodium malariæ, 247
Plegaphonia, 135
Pleura, diseases of, form of thorax in, 72
exploratory puncture of, 136
left, exudation in, dulness of halfmoon-shaped space in, 275
thickened, sensation of resistance over, 102
thickening of, diminished vesicular breathing in, 124
percussion-deadness in, 112
tumors of, weakness of vocal fremitus in, 134
Pleural cough, 142
exudations, chemical examination of, 139
determination of albumin in, 139
diminished vesicular breathing in, 124
examination of, for bacteria, 137
hemorrhagic, 139
Pleural exudations, microscopic examination of, 137
sacs, boundaries of, 71
sinus, complementary, 71
Pleurisy, asymmetry of breathing in, 81
carcinomatous, exudation in, 137
diaphragmatic, tympanitic sound in, 112
differential diagnosis from pericarditis and peritonitis, 200
diminution of motility of lungs in, 118
of spleen-dulness after, 301
downward displacement of liver in, 289
drawing-in of thorax from, 77
dyspnea in, 85
effect of, on position of liver, 295
encapsulated, 111
exudative, non-tympanitic sound in, 89
from fracture of rib, 90
lagging in, 91
leukocytosis in, 242
position in bed in, 29
streptococcus pyogenes in exudations of, 138
Pleuritic exudation a cause of deadening of percussion resonance, 111
anuria in, 367
feculent ichorous, micro-organisms in, 138
ichorous, micro-organisms of, 138
increased vocal fremitus in, 134
sensation of resistance over, 102
weakness of vocal fremitus in, 134
friction-sounds, 132, 200
Pleuritis, adhesive, vein-nets on chest and back in, 225
vital capacity of lungs in, 141
diaphragmatica, detection of, by palpation, 91
exudative, bronchial breathing in, 126
diminished inspiratory pressure in, 141
displacement of apex-beat in, 173
displacement of liver in, 291
extension of liver-dulness in, 295
tympanitic sound in, 98
exploratory puncture in, 136
Fränkel's pneumococcus in, 163
friction-sound in, 132
pulse in, 218
purulent, 90
sicca a cause of friction sounds, 132
staphylococcus pyogenes albus in the exudation of, 138
tenderness of thorax in, 90
with emphysema, friction-sounds in, 133
Pleximeter, 92, 93
Pneumatometry, 141
Pneumatoscope of Gabritschewsky, 135
Pneumococci, methods of staining, 163
Pneumococcus, Fränkel's, 162
Friedländer's, 162
in cerebrospinal fluid in epidemic cerebrospinal meningitis, 435
of Fränkel in pleural exudations, 138
Pneumonia, abnormally loud, deep percussion-sound in, 116
absence of cough a bad sign in, 165
asymmetry of breathing in, 81
bloody sputum in, 145
bronchial breathing in, 125

Pneumonia, catarrhal, crepitant râles in, 13
deadening of percussion-sound in, 110
thickening and deadening with, 110
caused by dust, 21
cocci in sputum, 162
course of fever in, 61
critical sweat in, 34
croupous, bloody sputum in, 146
bronchial breathing in, 126
crepitant râles in, 131
Curschmann's spirals in, 155
deadened resonance over lungs in, 109
feeling of resistance in, 109
fever in, 59
frequent respiration in, 84
hemorrhage from lungs in, 146
herpes facialis with, 145
one-sided expansion of chest in, 76
spiral casts in, 149
tenderness with, 90
tube-casts in, 149
tympanitic sound in, 98, 113
diminution of chlorids of urine in, 392
dry cough in, 143
dyspnea in, 85
epileptiform convulsions in beginning of, 490
epithelium in sputum of, 151
extended perception of heart-sounds in, 187
Fränkel's pneumococcus in sputum of, 162
frequent pulse in, 205
friction-sounds with, 133
Friedländer's pneumococcus in, 163
increase of urea in, 392
of vocal fremitus in, 134
lagging in, 91
leukocytosis, 242
lobular, thickening and deadening with, 110
massive, dulness in, 110
sensation of resistance in, 102
mucous threads in sputum of, 150
non-tympanitic sound in, 99
peptone in sputum of, 164
peptonuria with, 398
position in bed in, 29
pulse in, 205
ringing dry râles in, 129
smallness of halfmoon-shaped space in, 276
spirals in sputum of, 149, 150
sudden stopping of cough in, 142
tenderness of thorax in, 90
vomiting in, 326
Pneumonic deposits, cracked-pot sound in, 116
Pneumonokoniosis, pleuritic friction-sounds, 132
Pneumopericardium, diminution of heart-dulness in, 182
metallic heart-sounds in, 191
metallic sound over, 99
tympanitic sound in, 98
Pneumothorax a cause of cyanosis, 39
amphoric breathing in, 127
bronchial breathing in, 126
cracked-pot sound in, 116
displacement of apex-beat in, 173
downward displacement of liver in, 289
dyspnea in, 85
Pneumothorax, effect of, on position of liver, 295
expansion of chest in, 76
liver-dulness in, 295
loud, deep percussion-sound in, 117
lung-sound over, 99
metallic heart-sound in, 191
râles, 131
sound over, 98
non-tympanitic sound over, 99
one-sided expansion of lungs in, 117
plegaphonic sound in, 135
position in bed in, 29
pulse in, 211
relative liver-dulness in, 296
tympanitic sound in, 98, 116
weakeness of vocal fremitus in, 134
Poikilocytes, 240
Poikilocytosis, 241
Points douloureux, 448
of electrical stimulation, 469. See *Motor points*.
Poisoning, acute, albuminuria in, 394
disturbance of consciousness in, 437
Poisons in urine, 411
Poles of battery, how to distinguish, 466
Polio-encephalitis, choreic motions in, 492
Poliomyelitis, 454
acute, arrest of growth of bones after, 530
disunion of atrophy and paralysis in, 456
bladder disturbance absent in, 528
decreased tendon reflexes in, 461
epileptiform attacks in beginning of, 490
reaction of degeneration in, 484
vasomotor disturbances in, 526
Pollution from irritation of penis, 529
Polydipsia, polyuria from, 367
Polyesthesia, 443
Polypi, nasal, relation of, to nervous diseases, 525
Polyuria, 366
in nervous disease, 528
nervous, 367
Pons Varolii, symptom of disease of, 425
Portal vein, obstruction of, ascites in, 282
occlusion of, enlargement of spleen in, 298
Position of patient, 28
passive dorsal, 28
Positions, compulsory, 30
Posner on albumin in urine in health, 366
Post-epicritical increase of urea in pneumonia, 392
-epileptic coma, 490
Postponing intermittent fever, 63
Posterior rhinoscopy, 544
Posticus paralysis, 542
Posture of patient, 28
Pregnancy, double sound over crural artery in, 222
scars of, 46
Pressing noise, 268
Pressure-sense, testing of, 440
Presystolic heart-murmur, 194
Primary deviation, 515
myopathic atrophy, 455

Profession as a factor in causation of disease, 21
Progressive muscular atrophy, 454
cyanosis in, 39
fibrillary contractions in, 490
paralysis, diminution of temperature in, 525
fever in, 525
light rigidity of pupil in, 518
perforating disease of foot in, 530
Propepsin, secretion of, 321
by stomach, 305
Propeptone, test for, 322
Prostate, hypertrophy of, residual urine in, 368
Prostatitis, prostatorrhea in, 413
Prostatorrhea, 413
Prune-juice sputum, 145
Prussic-acid poisoning, glycosuria in, 401
odor from mouth in, 253
Pseudo-crisis, 61
Pseudobulbar paralysis, speech-disturbance rare in, 502
Pseudohypertrophy, 456
Pseudoleukemia, ascending venous pulse in, 229
Pseudoparalysis, spastic, 456
Pseudo-vacuoles, 249
Psoas, paralysis of, effect of, 500
Psychoses, increased secretion of saliva in, 527
Ptomaïns, connection between, and cystin in urine, 390
Ptosis from paralysis of oculomotorius, 515
Ptyalism, 256
Puerile breathing, 122
origin of, 122
Puerperal fever, peptonuria with, 398
temperature in, 57
pyemia, streptococcus pyogenes in, 549
Pulmonary artery, aneurysm of, 219
heart-sound, strengthened, 188
stenosis, effect of, 168
Pulsating tumor of spleen, 298
Pulsation, epigastric, 177, 272
Pulse, 201
anomalies of, in nervous disease, 526
as an indication of strength of heart, 218
auscultation of, 221
bulbar, 227
capillary, 220
-curve in health, 212
pathological, 213
daily variations in, 201
diagnostic value of, 216
dicrotic, 213
empty, 208
equal, 203
examination of, diagnostic value of, 216
filiform, 209
frequent, occurrence of, 204
full, 208
hard, 210
hardness of, 210
influence of deep breathing upon, 202
of external temperature upon, 202
of meals on, 202
of mental excitement upon, 202
of movement on, 202
Pulse, influence of position of body on, 202
of sex on, 201
of size of body on, 201
of sleep upon, 202
irregular, 209
large, 208
liver-, 221
method of observing, 202
monocrotic, 213
normal, 201
frequency of, 201
palpation of, 201
pathological frequency of, 203
quality of, 203, 208
quick, 209
radial, examination of, 201
in pericarditis, 174
regular, 209
retardation of, 203
rhythm of, 202
slow, 209
small, 208
sphygmography of, 211
subdicrotic, 213
superdicrotic, 213
suspended, 207
symmetry of radial, 210
tense, 210
thread-like, 209
trembling, 209
venous, 226
want of rhythm of, 207
wire, 210
Pulsus æqualis, 209
alterans, 209
bigeminus, 203
tracing of, 215
celer, 209
sphygmographic curve of, 214
deficiens, 207
dicrotus, 209
differens, tracing of, 215
durus, 210
inæqualis, 209
intercidens, 208, 209
intermittens, 203, 207
magnus, 208, 209
mollis, 210
paradoxus, 209
parvus, 208
plenus, 208
rarus, 203
tardus, 209
tracing of, 214
tensus, 210
tremulus, 209
trigeminus, 207
alterans, 203
vacuus, 208, 210
Punctiform ecchymoses in epilepsy, 529
Puncture, exploratory, of heart, 200
of liver, 292
lumbar, 434
of abdomen, to draw off ascitic fluid, 285
of peritoneal cavity, diagnostic value of, 285
of pleura, exploratory, 136
Pupil, contracted, 517

Pupil, dilatation of, 517
from paralysis of oculomotorius, 515
effect of poisons on, 517
inequality of, 517
light reflex of, 518
light rigidity of, 518
reflex changes in size of, 517
rigid, 518
rigidity of, as a symptom, 533
size of, 517
Purpura hæmorrhagica, cutaneous hemorrhage, 46
Purulent exudations in sputum, 143
pleuritis, 90
sputum, 145
stools, 339
Pus-cocci in urine, 385
-corpuscles in urine, 377, 378
in cerebrospinal fluid, 435
in larynx, 539
in stomach-contents, 315
in stools, 339
in urine, 377
makes it turbid, 371
odor of urine in, 373
reaction of urine with, 373
vomiting of, 330
Putrefaction of stomach-contents, 309
Pyelitis, bacterium coli commune in, 383
calculosa, thickening and distention of ureters in, 360
cylindroids in urine of, 375
white blood-corpuscles and pus in urine of, 377
Pyelonephritis, peculiar hyaline casts in urine of, 381
Pyemia, cutaneous hemorrhage in, 46
enlargement of spleen in, 298
frequent pulse in, 205
herpes in, 45
intermittent fever in, 63
pulse in, 205
pus-cocci in urine in, 385
retinal hemorrhage in, 548
staphylococcus pyogenes in, 548
streptococcus pyogenes in, 549
sweat in, 34
Pyemic deposits in lungs, friction-sounds in, 133
endocarditis, retinal hemorrhage in, 548
Pyloric stenosis. See *Stenosis of pylorus.*
Pylorus, congenital displacement of, 271
position of, 268
spasm of, 310
stenosis of, effects of, on digestion, 310
Pyonephrosis, palpation of, 358
Pyopneumocardium, metallic pericardial splashing in, 200
Pyopneumothorax, change of boundaries of dulness in, 112
metallic sound in, 117
splashing sound in, 133
subphrenicus, 284
Pyramidal tract, 416
above oblongata, affections of, speech-disturbance in, 502
effect of lesion of, 425
Pyramidal tract, lateral, 417
Pyrocatechin, effect of, in urine, 371
Pyrosis hydrochlorica, superacidity in, 325
Pyuria, 377
due to strongylus gigas, 382

QUADRICEPS muscles, paralysis of, effect of, 500
Qualitative irritability of muscles from galvanic stimulation, 478
Quantitative excitability of nerves and muscles, 476
Quartan intermittent fever, 63
Quincke on fever from injury of cervical spinal cord, 525
on progressive venous pulse, 229
on proportion between urine secreted during day and that secreted at night, 362
pseudo-vacuoles, 249
Quincke's lumbar puncture, 434
Quinia in urine, 411
Quotidian intermittent fever, 63

RACHITIC chest, 78
Rachitis as a cause of chest deformity, 79
diminution of phosphates in urine in, 392
enlargement of cranium in, 430
how distinguished from hydrocephalus, 431
Radial artery, abnormal course of, 208
nerve, cutaneous filaments of, supplying dorsal side of arm, 449
electric stimulation of, 472
muscles supplied by, electrical examination of, 475
paralysis of, 451
pulse. See *Pulse.*
asymmetry of, 245
Radomyski, casts in urine in disturbance of circulation, 380
Râles, bubbling, 131
consonant, 130
crepitant, 131
dry, 128
"hinted," 130
metallic, 128
moist, 129
palpation of, 89
ringing, 130
sibilant, 128
viscid-moist, 129
Raspberry-jelly sputum, 141
Reaction of degeneration, 478
as a symptom, 531
complete and partial, 479
course of, 480
mechanical, 485
mixed, 484
partial, significance of, 484
relation of, to course of paralysis, 481
schematic representation of, 479
significance of, 484
varieties of, 483
when absent, 484
when occurs, 484
of expectoration, 144
of Koch's tuberculin, 165

Recoil-elevation of pulse-curve, 212
Records of cases, value of, 24
Rectum, palpation of, 280
stenosis of, pain at stools in, 335
tumors of, examination of, 279
Rectus externus of eye, paralysis of, 515
inferior, paralysis of, effect of, 515
internus, paralysis of, effect of, 515
superior, paralysis of, effect of, 515
Recurrent fever, 64
spirillum recurrens in blood of, 246
paralysis, 542
Red blood-corpuscles. See *Blood-corpuscles.*
in sputum, 151
Redness of larynx, when occurs, 538
of skin, abnormal, 37
Reducing substances in urine in health, 366
Reflex centers, 419
cremaster, 458
of sole of foot, 457
patellar, 459
rigid pupil, 518
skin, testing of, 457
tendo-Achillis, 460
Reflexes, 457
abdominal, 458
fascial, 460
of mucous membranes, 458
periosteal, 460
skin, 457
tendon, 458
Reiss on strong percussion to determine size of heart, 179
Relapsing fever, 64
Relative deadness of sound, 95, 96
Remittent fever in the course of febrile diseases, 61
range of temperature in, 56
vomiting in, 326
Renal albuminuria, when occurs, 394
calculi, fever in, 57
casts, additions to, 381
kinds of, 380
colic, 391
engorgement, hyaline casts in urine of, 380
epithelium in urine, 379
Rennet ferment, secretion of, 306
zymogen in gastric juice, determination of, 321
Residence, place of, as a factor in cause of disease, 21
Residual urine, 368
Resistance of skin to electrical conduction, determination of, 474
sensation of, 102
Resonance, deadened, over the lungs, 109
when deadened, 109, 112
Resorcin as a test for hydrochloric acid in stomach, 317
in urine, 411
Resorption of exudation or fluid of edema, polyuria from, 366
of food, 335
Respiration, anomalies of, 79
Biot's, 83
Cheyne-Stokes, 81
examination of movements of thorax in, 91
Respiration in diseases of nose, 66
increased and forced, 86
normal, 72
auscultatory signs in, 121
rapidity of, 73
Respiratory apparatus, disturbances of, in nervous disease, 525
change of acme of sound, 115
motions, effect on circulation in jugular veins, 225
sound, change of, 99
sounds, pathological, 123
Retentio urinæ, 528
Retinal apoplexy a forerunner of cerebral hemorrhage, 547
arteries, pulsations of, 548
hemorrhages, 547, 548
in general hemorrhagic diathesis, 514
Retinitis albuminurica, 547
pigmentosa from syphilis, 547
syphilitica, 547
Retroperitoneal glands, enlargement of, 303
confounded with aortic aneurysm, 303
Retropharyngeal abscess a cause of cyanosis, 38
detection of, 260
Reuss' formula for determination of albumin, 139
Reynolds on test for acetone in urine, 409
Rhagades, significance of, 253
Rheostat, importance of, 462
Rheumatic facial paralysis, disturbance of hearing with, 523
paralyses, reaction of degeneration in, 484
Rheumatism, articular, entrance of, through tonsils, 258
perspiration in, 34
chronic articular, phosphaturia in, 389
pain in spine in, 447
of chest-muscles, pain of, 90
of thorax, frequent respiration in, 84
peptonuria with, 398
Rhinoscopy, 543
anterior, 543
posterior, 544
Rhomboideus, paralysis of, effect of, 497
Rhubarb, color of urine after taking, 371
Ribs, caries of, as a cause of pleurisy, 90
fractures of, diagnosis of, 90
increased respiration in, 84
pain in disease of, 90
Rice-water stools, 338
Richardson's sphygmograph, 211
Riegel's double stethograph, 141
Rigidity of muscles, 456
Ringing râle, 130
Risus sardonicus, 490
Robertson's pupil, 518
Rod-pleximeter percussion, 117
Rolando, fissure of, location of, 432
Rolando's line, 432
Romberg's symptom, 445
Rosenbach's modification of Gmelin's test for bile-pigments, 400
Rosenstein on absence of free hydrochloric acid from stomach in diabetes, 324
Roscola, 44

Roseola syphilitica, 44
Rosin's test for bile-pigments in urine, 401
Rössler, experiments with Esbach's albuminometer, 398
Round worms, 343
in vomit, 331
Rubor pudicitiæ, 37
Rumpf on signification of Wintrich's change of sound, 114, 115

Saccharomyces in urine, 385
Sahli on cyanosis in Cheyne-Stokes respiration, 82
on origin of puerile breathing, 122
Sahli's improvements of Penzoldt's test, 323
theory of anemic murmurs, 197
Salicylic acid in urine, test for, 410
Saliva, diminution of, 256
examination of, 256
increased secretion of, 256
in nervous diseases, 527
of nephritis, urea in, 256
Salivary glands, examination of, 256
Salivation, 256
Salkowski-Leube method of quantitative testing for sugar in urine, 404
Salkowski on composition of Charcot-Leyden's crystals, 157
Salol in urine, 411
method of Ewald for determining rapidity with which stomach empties its contents into duodenum, 322
Salter on displacement of lower border of lungs in lying down, 109
Sänger, palpation of ureters through vagina, 359
Santonin, color of urine after taking, 371
Sarcina pulmonalis in sputum, 159
ventriculi in vomit, 331
Sarcinæ in stomach-contents, 316
in urine, 385
Sarcoma of kidney, palpation of, 358
of larynx, 541
of skull, projections caused by, 431
Satyriasis, 439
Scanning speech, 502
Scaphoid drawing-in of abdomen, 279
Scarlet fever, condition of tonsils in, 259
cutaneous hemorrhage in, 46
enlargement of spleen in, 298
entrance of, through tonsils, 257
epileptiform convulsions in beginning of, 490
glycosuria in, 402
hemoglobinuria in, 370
mulberry-tongue in, 254
vomiting in, 326
Scars, 46
Scherwald's plegaphonia, 135
Schizomycetes in stomach-contents, 316
Schmidt discovered Curschmann's spirals in upper lobes of asthma-patients, 155
on Curschmann's spirals in hardened sputum, 155
Schmiedeberg's method of quantitative testing for sugar in urine, 405
Schultze on reaction of degeneration in myopathic muscular atrophy, 483
Sclerosis, amyotrophic lateral, 454
diminished electric irritability in, 485
increased tendon-reflexes in, 461
reaction of degeneration in, 484
arterial, "goose's throat" in, 220
multiple, allochiria in, 443
atrophy of optic nerve in, 513
concentric narrowing of field of vision in, 521
imbecility from, 438
intention tremor in, 489
narrowing of field of vision in, 521
nystagmus in, 516
primary atrophy of optic nerve in, 547
vertigo in, 438
of aorta, shown by stronger aortic second sound, 188
of arteries a cause of hypertrophy of left ventricle, 120
Scoliosis, 78
Scorbutus, cutaneous hemorrhage in, 46
hematuria in, 376
loosening of teeth in, 253
peptonuria with, 398
Scotoma, central, when occurs, 521
Scurvy, oxybutyric acid in urine, 408
Scybala, recognition of, 279
Secondary deviation, 515
Sediments in urine, 363, 365
pathological, 374
Sedimentator, Stenbeck's, 374
Sehrwald, method of determining urea of urine, 392
Seitz, metamorphosing breathing, 127
Self-registering thermometer, advantages of, 51
Semi-decussation of optic tract, 420
Seminal fluid, examination of, 411
Senator on albumin in healthy urine, 366
on ascending venous pulse in pseudoleukemia and leukemia, 229
on leukocytes in urine, 378
on origin of epithelium in sputum, 151
Senna, color of urine after taking, 371
Sensation of movements, testing of, 440
of resistance, 102
of spasm, testing of, 444
Sense of heat and cold, testing of, 440
of pressure, testing of, 440
of space, testing of, 440
of touch, testing of, 439
organs, 513
Sensibility, cutaneous, 439
delayed, 443
disturbances of, 439
double, 443
to pain, testing of, 441
Sensitive tracts, 419
Sensory aphasia, 506
Sepsis, enlargement of spleen in, 298
retinal hemorrhage in, 514
Seropneumothorax, change of boundaries of deadness in, 112
metallic sound in, 117
splashing in, 133

Serous sputum, 145
Serratus muscle, paralysis of, effect of, 497
Serum-reaction in typhoid fever, 247
Shaking-spasm, 489
Sharpness of vision, testing of, 519
Shoemakers' breast, 79
Shreds of tissue in urine, 379
Sibilant râles, 128
Sibson's furrow, 69
Siegel's pneumatic ear-speculum, 546
Sieveking's esthesiometer, use of, 440
Sight, center and tract for, 420
Silver in urine, 411
Simon, palpation of ureter through the rectum, 359
Singing, loss of power of, in atactic aphasia, 508
Singultus, 496
Sinus head, 342
 phrenico-costalis, 71
Situs viscerum inversus, 167
Sjöqvist's method of determining free plus loosely-combined hydrochloric acid in stomach, 319
Skeleton, defective development of, 30
Skin, abnormal redness of, 37
 appearance of, in ascites, 282
 blue-red, 38
 bronze, 43
 color of, 34
 disturbances of, relation to nervous disorders, 529
 edema of, 46
 emphysema of, 49
 gray, from silver deposit, 44
 hemorrhages in, 45
 moisture of, 32
 nutrition of, 32
 pale, 35
 -reflexes, 457
 mechanism of, 458
 state of nutrition of, 32
Skoda, influence on development of auscultation, 119
 on percussion-sounds, 96
 recoil-theory of apex-beat, 172
 reference to, as reviver of percussion, 92
Skull, asymmetry of, 431
 examination of, 430
 form of, 431
 projections and depressions on, 431
Sleep-paralysis, effect of, 498
Small-pox, cutaneous hemorrhage in, 46
 enlargement of spleen in, 298
 laryngitis in, 68
 vomiting in, 326
Smell, loss of sense of, 524
 testing of, 523
 tract of, 421
Snellen's plate, 519
Soft palate, electric stimulation of, 471
 innervation of, 494
 paralysis of, 494
Solidification of lungs, percussion-sound in, 101
Sommerbrodt's sphygmograph, 211
Somnolence, definition of, 437
Sopor, definition of, 437
Sound-image center, location of, 505
 metallic, 98
 use of, in examining nasal cavities, 544
Sounding of the esophagus, 264
 of stomach, 272
Sounds of falling drops, 131
 percussion, 94
Space-sense, testing of, 440, 445
Spasm of chest-muscles, dyspnea in, 85
 of glottis a cause of cyanosis, 38
 of muscles of larynx, 541
 of respiratory muscles a cause of cyanosis, 39
 -sense, testing of, 444
 shaking-, 489
Spasms, clonic, 488
 when occur, 490
 co-ordinated, 491
 dissociated, 490
 kinds of, 489
 of voluntary muscles, 488
 tonic, 488
 when occur, 490
Spastic paralyses, 419
 pseudoparalysis, 456
 spinal paralysis, increase of tendon-reflexes in, 461
Specific gravity of urine, 363
 in disease, 372
Speech-center, location of, 505
 motor, location of, 505
 disturbances of, 502
 how acquired, 503
 scanning, 502
Sperma-crystals in semen, demonstration of, 412
Spermatic fluid, abnormal, 412
 normal, 411
Spermatorrhea, 414
Spermatozoa in urine, 379
Sphygmograph, pathological forms of pulse shown by, 212, 213
Sphygmography, 211
Sphygmomanometer, 210
Spinal column, diminished mobility of, 434
 form of, 434
 sensitiveness to pressure, 434
 cord diseases, localization of, 427
 feeling of constriction in, 446
 topographical diagnosis of, 423
 relation of, to spinal column, 434
 disease, aspermatism from, 412
 spermatorrhea in, 414
 irritation, pain in spine in, 447
 sensitiveness to pressure in, 434
 paralysis. See *Paralysis.*
Spirals, Curschmann's, 154
 in expectoration of bronchial asthma and croupous pneumonia, 149
 in sputum, 154
Spirillum recurrens in blood, 246
 in urine, 385
Spirometry, 141
Spleen, anatomy of, 296
 auscultation of, 302
 consistence of, 298

Spleen, displacement of, 299
-dulness, 299
diminution of, 301
enlargement of, 301
size of, 300
enlargement of, diagnosis of, 301, 302
expansion of thorax in, 77
from general venous engorgement, 224
projection of left hypochondrium in, 297
smallness of Traube's space in, 275
examination of, 296
form and surface of, 298
inspection of, 297
-lung angle, 297
mobility of, 299
palpation of, 297
pathological relations of, 301
percussion of, 299
peritoneal friction-sounds heard over, 302
relation to colon, 299
size of, 298
systolic pulsation of, 298
tenderness of, diseases in which it occurs, 298
tumors of, 298
wandering, 298, 299
when it can be felt, 298
Spontaneous pain, 446
Sputa, confluent, 145
Sputum, 143
actinomyces in, 164
albuminous corpuscles in, 164
animal parasites in, 157
aspergillus in, 164
bile-pigment in, 147
bloody, 145
cartilage in, 148
cercomonas in, 158
Charcot-Leyden's crystals in, 156
chemical examination of, 164
cholesterin-crystals in, 156
coal-soot in, 147
crystals in, 149, 155
crystals of fatty acid in, 156
distoma pulmonale in, 158
echinococcus in, 157
elastic fibers in, 152
epithelial cells in, 151
ferment in, 164
fibrinous tubes in, 148
foreign substances in, 147
Fränkel's pneumococcus in, 162
Friedländer's pneumococcus in, 162
fungi in, 149, 158
green, 147
heart-disease cells in, 151
hematoidin in, 147
infusoria in, 158
iron-dust in, 147
leptothrix buccalis in, 158
lung-tissue in, 147
microscopical examination of, 150
method of, 153
monas in, 158
mould in, 164
mucopurulent, 145
Sputum, mucous corpuscles in, 150
mucus in, 144
odor of, 147
oxalate of lime in, 157
peptone in, 164
Pio Foà's coccus in, 162
pneumonia cocci in, 162
prune-juice, 145
purulent, 145
red blood-corpuscles in, 151
sarcina pulmonalis in, 159
serous, 145
starch-corpuscles in, 155
threads of muscles in, 150
triple phosphate in, 157
tubercle bacillus in, 159
tyrosin in, 157
white blood-corpuscles in, 150
Square position of liver, 295
Squibb's modifications of urinometer, 364
Squirting noise, 268
Stadelmann on ferment in sputum of phthisis, 164
on formation of bile-pigment in poisoning, 41, 42
on oxybutyric acid a cause of diabetic coma, 407, 408
on peptone in sputum, 164
Stagnation of stomach-contents, 309
Staphylococcus pyogenes albus, 548
in exudation of empyema, 138
in pleural exudates, 138
aureus, 548
in exudations of empyema, 138
cereus, 548
citreus, 548
in exudations of empyema, 138
importance of, 548
Starch-corpuscles in sputum, 155
-grains in vomit, 331
Starches, digestive changes in, 304
Stenbeck's sedimentator, 374
Stenosis, aortic, effect of, 168
pulse in, 217
-curve in, 214
pulsus tardus in, 210
systolic heart-murmur over carotid in, 222
arterial, pulsus tardus in, 210
intestinal, distention of abdomen in, 278
laryngeal, distinguished from tracheal stenosis, 67
mitral, arrhythm in, 207
diastolic murmur in, 193
division of second apex-sound in, 190
effect of, 168
irregular pulse in, 209
pulse-curve in, 215
pulse in, 216
small pulse in, 209
strengthening of apex-sound in, 188
of air-passages, diminished vesicular breathing in, 124
breathing in, 81
of aorta, commencement of absence of apex-beat in, 175
systolic murmur in, 193

Stenosis of bronchi, inspiratory drawing-in of, 87
of esophagus, symptoms of, 263
of heart-openings, murmurs in, 191
of intestine, obstipation in, 334
visible peristalsis in, 279
of larynx, inspiratory drawing-in of, 87
of pylorus, carcinomatous, effect on digestion, 310
effect on digestion, 310
hypacidity in, 324
of rectum, pain at stools in, 335
of trachea, inspiratory drawing-in of, 87
of valve, 168
ostii aortæ, retardation of pulse in, 204
pulmonary, systolic murmur in, 193
effect of, 168
pyloric, anacidity of stomach in, 325
diminished volume of abdomen in, 278
food in stomach after digestion of test-breakfast, 323
tricuspid, diastolic murmur in, 193
effect of, 168
venous engorgement in, 224
Stereognosis, 446
Sterility, 21
Sternal lines, 69
Sternum, percussion-sound over, 106
Stethography, 141
Stethoscope, varieties and use of, 119
Stiffness of joint, diminution of power of motion from, 452
Stigmates hystériques, 491
as a symptom, 533
Stintzing on quantitative excitability of nerves and muscles, 476–478
on reaction of degeneration, 483
Stokes on Cheyne-Stokes respiration in fatty degeneration of heart, 82
on vesicular murmur louder on left side than on right, 122
Stolnikow on ferment in sputum, 164
Stomach, absorption by, 307
anacidity of, 309
anatomy of, 268
antiperistalsis of, 272
artificial distention of, 271
emptying of, 303
artificially dilated, percussion of, 274
auscultation of, 276
boundary illustrated, 274
cancer of, tumors in, 273
catarrh of, stomach-contents in, 311
-colon fistula, diagnosis of, 280
-contents, abstraction of, for examination, 312
examination of, 303, 315
by microscope, 316
by the eye, 315
chemical, 316
fermentation of, 309
gases in, 310
putrefaction of, 309
removal of, for examination, 303
stagnation of, 309
-cough, 142
diaphanoscopy of, 276
-digestion described, 304
Stomach-digestion, duration of, 305
of nurslings, 308
results of examination of, 323
dilatation of, from pyloric stenosis, vomiting in, 327
-disease, coating of tongue in, 255
vertigo from, 438
distensibility of, 271
distention of, 271
ectatic dilatation of, 310
examination of, 268
during digestion, 277
-gases, determination of, 314
greater curvature of, 269
-heart space, 275
hour-glass, recognition of, by inflation, 272
how soon it empties its contents into duodenum, 322
hypacidity of, 308
hyperacidity of, 309
hypertrophy of, peristaltic motions in, 272
illumination of, 276
increased resistance of, on palpation, 272
inflated, metallic heart-sounds in, 191
inflation of, for diagnosis, 271
inspection of, 270
length of time that food remains in, 311
lesser curvature of, 269
-liver space, circular, 275
location of, 268
-lung space, circular, 275
metallic sound over, 98
motor activity of, diminution of, 309
non-tympanitic sound over, 99
palpation of, 270
by striking, 272
percussion of, 273
with rod-pleximeter, 275
peristaltic motions in, 272
peristaltic unrest of, 272
-sound, contraindications to use of, 312
introduction of, 313
after test-meal, 315
sounding of, 272
-splashing, 276
subacidity of, 308
superacidity of, 309
tenderness of, to palpation, 273
-tubes, 313
tumors of, dulness on percussion, 275
tympanitic sound over, 98
washing out of, 312
Stomatitis, bacteria of mouth increased in, 257
enlargement of tongue in, 254
Stone in bladder, hematuria in, 376
in kidney, diminished secretion of urine in, 368
tenderness with, 357
Stools, additions to, from alimentary canal, 348
admixtures of, 336
amount of, 335
bilious, 337
bloody, 338
color of, 336
constituents of, 336

Stools, consistence or form of, 336
fatty, 338
foreign bodies in, 337
mucous, 337
odor of, 336
pain with, 335
purulent, 339
reaction of, 336
rice-water, 338
watery, 338
Strabismus, 514
convergent, 514
divergent, 514
Strangulation of intestine, obstipation in, 334
Strangury from concentration of urine, 361
Streptococcus erysipelatis, 549
pyogenes, 549
in exudations of empyema, 138
in pleural exudations, 138
Stricture, aspermatism from, 412
of esophagus, location of, by sounding, 266
of urethra, diminished quantity of urine in, 368
residual urine, 368
Stridor laryngeus, 67
vel trachealis, 87
Strongylus gigas causes pyuria and hematuria, 382
Structure of the body, 30
Struma, murmurs over thyroid glands in, 223
Strümpell on associated movements in spastic spinal paralysis, 492
on choreic motions in polioencephalitis, 492
on uremia, 399
perverse sensibility to changes of temperature, 443
Strychnia in urine, 411
-poisoning, increase of tendon-reflexes in, 461
Stupor, 437
Subacidity of stomach, 308
Subclavian artery, auscultation of, 221
left, compression of, pulsus differens in, 215
Subdicrotic pulse, 213
Subjective sensations of vision, 522
sensibility of hearing, 523
Subphrenic abscess, 284
downward displacement of liver in, 289
Subscapularis, paralysis of, effect of, 498
Succussion, Hippocratic, 133
Sudamina, 45
Sugar, absorption of, by stomach, 307
in cerebrospinal fluid, 435
in urine, approximate determination of, 406
determination of, by circumpolarization, 406
in health, 366
qualitative tests for, 402
quantitative tests for, 404
Sulphuretted hydrogen in urine, 373
Sulphuric acid in urine, 392
poisoning, glycosuria with, 401
Superacidity of stomach, 309
Superdicrotic pulse, 213
Supinator longus, electric stimulation of, 471
Suppuration, fever in, 57
Supraclavicular depressions in emphysema of lung, 75
percussion of, 104
point, 471
Supra-orbital nerve, distribution of, 448
Suprascapular space, 69
Supraspinatus muscle, function of, 497
Suspended pulse, 207
Swallowing, normal sounds of, 268
Sweating, local, 34
Swelling of larynx, when occurs, 538
Syllable-stumbling, 507
Sylvian line, 432
Sylvius, fissure of, location of, 432
Symptoms of diseases of brain and spinal cord, 423
Syncope, 438
Syphilis as a cause of nervous disease, 529
azoöspermia in, 413
changes in eye in, 547
congenital, Hutchinson's teeth in, 253
enlargement of liver in, 289
hemoglobinuria in, 370
hereditary, lips in, 253
laryngitis in, 68
mucous membrane of mouth in, 255
neuralgia in, 447
nocturnal headaches in, 447
of central nervous system, light rigidity of pupil in, 518
scars on larynx from, 540
secondary, palate and tonsils in, 258
tertiary, palate and tonsils in, 258
tongue in, 255
ulceration of larynx in, 539
Syphilitic infiltration of larynx, 540
ulceration of larynx, 539
Syringomyelia, deforming arthritis in, 531
Syringomyelitis, trophic disturbances of bones in, 530
ulcerations in, 530
System-disease, 532
Systole, movement of blood in, 108
Systolia alterans, 175
Systolic drawing-in near apex of heart, 175
of intercostal spaces, 177
pulsation in epigastrium, 177
of spleen, 298
trembling in epigastrium, 177

Tabes, allochiria in, 443
ataxia in, 488
atonic paralysis in, 457
bladder crises in, 528
disturbances in, 528
brittleness of bones in, 530
cystitis in, 528
decline of genital function in, 529
delayed sensibility in, 443
dorsalis, atrophy of optic nerve in, 513
decreased tendon-reflexes in, 461
myosis in, 517
neuralgic, lightning-like pains in, 447
Romberg's symptom in, 445
feeling of constriction in, 446
gastric crises in, 527

Tabes, herpes zoster in, 529
inequality of pupils in, 517
intestinal crises in, 527
light rigidity of pupil in, 518
migraine in, 447
mydriasis in, 517
pain in spine in, 447
partial paralysis of pressure-sense in, 440
perforating disease of foot in, 530
primary atrophy of optic nerve in, 547
rectal crises in, 527
residual urine in, 368
sensitiveness of spine to pressure in, 434
spermatorrhea, 414
Tachycardia, 203
occurrence of, 204
paroxsymal, 207
Tachycardic fit, 207
Tænia cucumerina, 343
mediocanellata, 342
saginata, 342
solium described, 341
eggs of, described, 341
Talma and Baas, explanation of moist râles, 129
Tape-worm, 341
Taste, loss of sense of, 524
testing of, 524
tract of, 421
Teale's case of high fever, 55
Teeth, caries of, in diabetes mellitus, 253
diseased, a cause of dyspepsia, 253
eruption of, a cause of disturbance, 254
examination of, 253
first, eruption of, a cause of disturbance in infants, 254
Hutchinson, 253
loosening of, 253
Temperature, daily variations of, 53
diagnostic value of, 57
elevated, 54
exacerbation of, in fever, 56
febrile, difference of, from normal, 55
highest recorded, 55
local elevation of, 65
local lowering of, 65
lowest observed, 56
methods of taking, 50
of the body, 50
normal, 53
perverse sensibilitity to, 443
remission of, 56
-sense, testing of, 440
subnormal, 56
Wunderlich's table of, 55
Tenderness in region of stomach, 273
of liver, 290
over kidneys, 357
Tendo-Achillis reflex, 460
Tendon-reflexes, 458
mechanism of, 461
Tenesmus, 335
of bladder, 361
Tensor of vocal cords, paralysis of, 543
Tensor tympani, predominant development of, disturbance of hearing in, 523
Teres minor, function of, 498
Tertian intermittent fever, 63
Test-meal, 308
composition of, 311
Ewald's, 311
Fleiner's, 312
Kussmaul's, 312
of Leube, 312
of meat and starches, 312
use of, 311
Tetanus, cyanosis in, 39
facial phenomenon in, 485
fever in, 525
increase of tendon-reflexes in, 461
increased irritability in, 485
mechanical excitability of nerves in, 485
opisthotonos in, 434
tonic spasms in, 490
Thallin in urine, test for, 411
Thermesthesiometer, 441
Thermometers, methods of using, 51
scale of, 51
selection of, 51
Thermometric scales, comparison of, 55
Thermopalpation, 92
Thigh-sound, 94
when heard, 99
Thoma-Zeiss counting-apparatus, 238
Thomsen's disease, 456
Thoracic muscles, paralysis of, 496
spasms of, 496
tonic and clonic spasms of, 496
Thorax, deformity of, a cause of displacement of apex-beat, 172
drawing-in or shrinking of one side of, 77
emphysematous, 75
form of, importance of, 30
inflated, 75
inspection of, 72
local expansions of, 77
measuring of, 140
normal form of, 72
one-sided expansion of, 76
pain caused by pressure on, 88
palpation of, 89
paralytic, 76
pathological forms of, 75
percussion of, 92, 103
phthisical, 76
testing the movement of, during respiration, 91
topographical anatomy of, 68
Thorn-apple crystals in urine, 389
Thought, examination of power of, 437
Threads, gonorrheal, in urine, 375
of mucus in sputum, 150
Thrill, hydatid, 292
Thrombosis, venous, 229
Thrush, coating of tongue by, 255
-fungus, recognition of, 257
-spore in feces, 349
Thumb, movements of, 499
Thyreo-arytænoideus internus, 541
Thyroid gland, murmurs over, 223
Tinnitus aurium, 523
Tissier on formation of urobilin in liver, 43
Tobacco amblyopia, appearance of fundus of eye in, 548

Tobacco amblyopia, central scotoma in, 521
Toluylendiamin, poisoning by, causes hemato-hepatogenous icterus, 401
Tongue, atrophy of, 494
circumscribed swelling of, 254
coating of, 255
color of, 254
dryness of, 254
electric stimulation of, 471
enlargement of, 254
examination of, 254
hardness of, 254
mulberry, 254
-muscles, functions of, 494
paralysis of, 494
scars on, 254, 255
trembling of, 254
wounds of, 254
Tonic spasms, 488
when occur, 490
Tonsil, abscess of, 259
ulcers of, 259
Tonsillitis, follicular, tonsils in, 258
tonsils in, 258
Tonsils, a seat of origin of septicopyemic disease, 258
entrance of infectious diseases by, 257
hypertrophied, 258
in diphtheria, 258
syphilitic scars on, 258
Tonus of paralyzed muscles, 456
Toothache from disease of the ear, 523
Topography of abdomen, 268
Torula cerevisiæ in vomit, 331
Touch, sense of, testing of, 439
Toxic paralyses, reaction of degeneration in, 484
Trachea, casts of, 149
normal percussion-sound over, 105
tympanitic sound over, 97
Tract of deep sensibility, 420
Tracts, centripetal, 419
motor, 416
sensitive, 419
Transition breathing, 127
Trapezius, paralysis of, effect of, 496
Traube, attempt to explain Cheyne-Stokes respiration, 83
reference to, as an improver of percussion, 92
sound over crural artery in aortic insufficiency, 122
theory of causation of moist râles, 129
Traube's halfmoon-shaped space, 270, 275
Traumatic hysteria, concentric narrowing of field of vision in, 521
neurosis, concentric narrowing of field of vision in, 521
Trautmann's pneumatic ear-speculum, 546
Treatment, effect of, determined by action of pulse, 218
Trembling, 489
Tremor, 489
alcoholic, 489
intention, 489
mercurialis, 489
saturninus, 489
senilis, 489
Trichina spiralis, 345
Trichinosis, 345
cyanosis in, 39
frequent respiration in, 84
Trichocephalus dispar, 345
Trichomonas vaginalis in urine, 383
Tricuspid insufficiency, relative, failure of pulmonary heart-sound in, 188
stenosis, effect of, 168
valve, auscultation of sound of, 184
Trigeminus, course of, 418
Triple phosphate in sputum, 157
in urine, 389
Trismus, tonic spasms in, 490
Trommer's test for sugar in urine, 402
Tropäolin reaction, 317
Tubercle bacilli, 552
in blood, 246
in feces, 353
in sputum, 159
microscopical demonstration of, 159
in urine, 383
staining of, 160, 161
of choroid, 547
Tubercular deposits, tympanitic sound over, 113
ulceration of larynx, 540
Tuberculosis, acute miliary, enlargement of spleen in, 298
frequent pulse in, 205
azoöspermia in, 413
casts in urine in, 380
Charcot-Leyden crystals in, 157
choroidal tubercle in, 547
color of face in, 37
deepening of supraclavicular depressions in, 73
dicrotic pulse in, 213
dyspnea in, 85
elastic fibers in sputum of, 152
fever of, 57
fibrin in urine with, 398
hemorrhage from lungs in, 146
hemorrhagic exudation in, 139
intestinal, pain on palpation of intestines in, 277
miliary, pleuritic friction-sounds in, 132
of apex of lung, altered vesicular breathing in, 124
resonance in, 110
of brain, poor nutrition in, 525
of intestine, Charcot's crystals in feces of, 349
of larynx, 68
swelling in, 538
of lungs, remittent fever in, 61
shown by tubercle bacillus, 162
of lymphatic glands, absorption of fats in, 336
of palate and pharynx, bacteriological determination of, 260
of pericardium, friction-sound in, 199
of urinary apparatus, shreds of tissue in urine of, 379
thickening or tenderness of ureters in, 360
pulmonary, Curschmann's spirals in, 155

Tuberculosis, pulmonary, hydrochloric acid in stomach in, 324
red border upon gums in, 254
shrinking of omentum in, 302
tubercle of choroid in, 547
tympanitic sound in, 98
ulceration of larynx in, 539
urogenital, tubercle bacilli in urine of, 383
Tuberculous infiltration and ulceration of larynx, paleness of mucous membrane in, 537
Tumors, expansion of thorax in, 77
dyspnea from, 88
in lung, percussion-sound of, 101
in region of stomach, 272
melanotic, color of urine from, 371
neuralgia from pressure of, 447
of bladder, hematuria in, 376
of brain, anosmia from, 524
catalepsy rare in, 492
central vomiting in, 326
choked disk in, 547
disturbance of consciousness in, 437
imbecility from, 438
migraine in, 447
vertigo in, 438
of chest, dyspnea in, 85
-cavity a cause of cyanosis, 39
sensation of resistance over, 102
tympanitic sound in, 112
of chest-wall, diminished vesicular breathing in, 124
of intestine, palpation of, 279
percussion of, 280
of kidney, detection of, 357
diagnosis of, from ovarian tumor, 358
differential diagnosis of, 359
hematuria in, 376
movable, 358
percussion of, 358
tenderness over, 357
of larynx a cause of cyanosis, 38
of liver change form of liver, 291
dulness in, 295
of lungs, percussion-sound in, 110
raspberry-jelly sputum in, 146
of meninges, sugar in cerebrospinal fluid in, 435
of rectum, examination of, 279
of spleen, 298
pulsating, 298
of stomach, dulness on percussion, 275
of vertebræ, sensitiveness of spine to pressure in, 434
within chest, bronchial breathing in, 125
Turban (C.) on curves of fever in phthisis, 62
Türck's reflector for examining larynx, 535
Turpentine, odor of urine after taking, 373
-poisoning, glycosuria in, 401
Tympanites, distention of abdomen in, 278
Tympanitic deadened sound, 113
sound, 94
change of pitch of, 114
closed, 97
conditions in which it is heard, 97
dull, 95
open, 97
Tympanitic sound over stomach, 273
pathological occurrence of, 112
what determines the pitch of, 97
Typhlitis, pain on palpation of right iliac fossa, 277
resistance in neighborhood of cecum in, 283
Typhoid bacilli in urine, 385
fever, bacilli of, 351, 550
bacillus of, in blood, 246
bloody stools in, 339
Charcot's crystals in feces of, 349
Cheyne-Stokes respiration in, 83
course of temperature in, 59
crystals of ammoniaco-magnesian phosphate in stools of, 349
dicrotic pulse in, 213
diminution of white blood-corpuscles in, 242
distention of abdomen in, 278
disturbances of consciousness in, 437
enlargement of spleen in, 298
entrance of, through tonsils, 258
frequent pulse in, 205
glycosuria in, 402
headache in, 447
hemoglobinuria in, 370
incontinentia alvi in, 528
leucin and tyrosin in urine of, 390
pain on pressure over intestines in, 277
peptonuria with, 398
position of children in bed in, 29
roseola in, 44
serum-reaction in, 247
spermatozoa in urine of, 379
stools in, 336
trembling of tongue in, 254
ulceration of larynx in, 539
Widal test for, 247, 551
Typhus abdominalis. See *Typhoid fever.*
bacillus, 550
fever, cutaneous hemorrhage in, 46
roseola in, 44
slow, 60
Tyrosin in sputum, 157
in urine, 390

Uffelmann on determination of lactic acid in stomach, 318
Uffelmann's test, 318
Uhthoff on light rigidity of pupil in syphilis of central nervous system, 518
Ulcer of stomach, bloody feces in, 339
blood in stools of, 329
bloody vomit in, 329
hypersecretion in, 325
normal digestion in, 324
perforating, hyperacidity in, 325
superacidity in, 325
white blood-cells in stomach-contents in, 316
Ulceration of large intestine, bloody stools in, 338
purulent stools in, 339
of larynx, 539
and trachea, emphysema in, 49
syphilitic, 539
tubercular, 540

Ulceration of rectum, odor of stools in, 336
of trachea and bronchial tubes, cartilage in sputum in, 148
Ulcers of intestine, tuberculous, tubercle bacilli in feces of, 353
Ulcus rotundum, hyperacidity in, 325
Ulnar nerve, electrical stimulation of, 472
Umbilicus, obliteration of, in ascites, 282
Undefined breathing, 127
Undigested food in stools, 336, 337, 347
Ungar on oxalate of lime in sputum of asthma, 157
Unilateral hypoglossal paralysis, 494
Unverricht, systolia alterans, 175
Urate of ammonium in urine, 389
of sodium and lime as a sediment in urine, 387
Urea in saliva of nephritis, 256
increase of, 392
normal amount of, in urine, 365
Uremia, 399
central vomiting in, 326
Cheyne-Stokes respiration in, 83
coma from, 437
diminution of urea in, 392
dyspnea in, 525
epileptiform spasms in, 490
headache in, 447
retinitis albuminurica in, 547
symptoms of, 399
Uremic coma, 399
Ureters, examination of, 359
obstruction of, diminished quantity of urine in, 368
palpation of, 359
Urethra, burning in, from concentration of urine, 361
Urethritis, white blood-corpuscles and pus in urine of, 377
Urethrorrhœa ex libidine, 413
Uric-acid concretions in urine, 391
crystals in urine, 386
Uric acid hinders examination for sugar, 365
in blood, 250
of urine, 365
test for, 381
when increased, 392
Uridrosis, 34
Urina nervosa, 367
spastica, 367
Urinary apparatus, examination of, 355
relation of disturbance of, to nervous diseases, 528
concretions, 391
testing of, 391
constituents in solution, 365, 391
sediments, 363, 365
examination of, 374
inorganic, 385
of organic bodies, 375
pathological, 374
Urination, frequent, from concentration of urine, 361
Urine, abnormal constituents of, 393
acidity of, determination of, 373
amphoteric reaction of, 365
as affected by medicines and poisons, 410
Urine, bacteria in, 361
blood-corpuscles in, 365
color and transparency of, 363
in disease, 368
concentrated, appearance of, 362
effects of, 361
concretions in, 391
daily average amount of, 362
diminished amount of, 367
discoloration of, 368–371
examination of, 360
hydrothionic, 373
incontinence of, 528
increased amount of, 366
involuntary passage of, when occurs, 528
manner of passing, 361
micro-organisms in, 361
mixed with feces, odor of, 373
mode of procedure in examining, 361
normal, 362
odor of, 365
due to medicines, 373
pathological, 373
pathological, 366
reaction of, 364
in disease, 372
residual, 368
retention of, 528
should be drawn with catheter when there is unconsciousness, 362
specific gravity of, 363
determination of, 364
in disease, 372
spectroscopic examination of, for blood, 400
Urinometer, 364
Urobilin, abnormal amount, in urine, 369
-icterus, 42, 369
in urine, 42
of fever, 369
tests for, 370
of urine, 362
Uva ursi, color of urine after taking, 371

VACUOLES, 249
Vagi, compression of, recurrent paralysis with, 543
Vagus, irritation of, retardation of pulse in, 204
Valleix's points, 448
Valve, stenosis of, 168
Valvular disease, combined, pulse in, 217
of heart, hematuria in, 375
insufficiency and its effects, 168
Varices, 46
Variola, leucin and tyrosin in urine in, 390
ulceration of larynx in, 539
Vasomotor disturbances in nervous diseases, 526
Vegetable parasites in stools, 349
in urine, 383
Veins, auscultation of, 230
examination of, 223
increased fulness of, 223
inspection and palpation of, 223

Veins of neck, differential diagnosis of different kinds of pulse in, 228
undulations in, 226
phenomena of circulation in, 229
Venous engorgement, causes of, 224
enlargement of spleen in, 298
humming, 230
liver-pulse, 228
pulse, 226
ascending, 229
double positive, in hemisystole, 229
positive, tracing of, 227
progressive, 229
systolic, 229
tracing of, 226
stasis, bloody vomit in, 329
thrombosis, 229
Ventricle, left, hypertrophy and dilatation of, shown by displacement of apex-beat, 173, 174
hypertrophy of, large pulse in, 208
hypertrophy of, shown by strengthened sound of corresponding valve, 188
Vermiform process, effect of lesions of, 427
"Verschlusszeit," 172
Vertebral canal, puncture of, 434
column. See *Spinal column.*
Vertigo, 438
ab aure læsa, 438
a stomacho læso, 438
of the eye, 514
Vesicular breathing, 121
alterations of, 124
special peculiarities of, 123
Vessels supplying brain, 421
Vicarious emphysema, 76
Vierordt (H.), method of measuring strength of heart-sounds, 188
on slowed coagulation of blood in chronic disturbances of nutrition, 250
(K.), originator of sphygmography, 211
Virchow on nature of epithelium in sputum, 151
Vision, field of, concentric narrowing of, 521
testing of, 520
sharpness of, testing of, 519
subjective sensations of, 522
Visual ammesia, 506
Vital capacity, 141
Vocal cords, movements of, in phonation, 537
position of, in paralysis, 542, 543
fremitus a means of distinguishing between pneumonia and pleuritic exudation, 134
testing for, 89
variations of, 134
weakness or suppression of, 134
Voice, alteration of, in disease, 67
auscultation of, 133
Voluntary muscles, innervation, function, and diseases that disturb them, 492
spasms of, 488
Vomicæ, tympanic sound over, 113
Vomit, bilious, 328
bloody, 328, 329
chemical examination of, 327
coffee-ground, test for hemin in, 330
Vomit, color of, 328
examination of, 327
macroscopical appearance of, 327
microscopical examination, 331
mucous, 328
corpuscles in, 331
odor of, 332
quantity of, 327
reaction of, 332
round worms in, 331
starch-grains in, 331
watery, watery-mucous, and mucous, significance of, 328
Vomiting, act of, described, 326
central, diseases in which it occurs, 326
fecal, 330
frequency of, 327
in brain disease, 527
in diseases of abdominal organs other than the stomach, 326
kinds of, clinically distinguished, 326
low specific gravity of urine in, 372
of blood, 328, 329
of pus, 330
phenomena associated with, 326
reflex, 326
time when it begins, 327
Vomitus matutinus potatorum, 327
of drunkards, 327
Von Basch on cause of dyspnea, 86
Von Frey on "recoil" of pulse-nerve, 212
sphygmograph of, 211
Von Jaffé, test for indican in urine, 369
Von Jaksch on alkalescence of blood in diabetes, 250
on auto-intoxication with acetonuria, 409
on diaceturia, 407
on phenyl-hydracin test for sugar, 403
on prognostic value of leukocytosis in pneumonia, 242
on starch-corpuscles in sputum of pulmonary gangrene, 155
on urobilin-icterus, 42
"vacuoles," 249
Von Jaksch's modification of Sjöqvist's method, 319
Von Limbeck on proportion of white blood-corpuscles to red ones, 242
Von Mering on absorption by stomach-walls, 307
on secretion of water by stomach, 307
Von Noorden on glycosuria, 402
Von Ziemssen, laryngitis hypoglottica, 538
on distention of stomach with carbonic acid, 272
on relations of size of body to vital capacity, 141
pulsus differens, 215
Von Ziemssen's method of obtaining blood for examination, 251

Wagner (E.), described peculiar sputum from hysteria, 146
Waldenburg's pneumatometer, 141
Wandering kidney, 357
left kidney, differentiation from wandering spleen, 359

Wandering liver, 289
absence of liver-dulness in, 296
right kidney, differentiation from distended gall-bladder or echinococcus, 359
spleen, 298, 299
Wasting diseases, heart-murmurs in, 197
Water, absorption of, by stomach, 307
-brash of drunkards, vomiting in, 328
whistling, 131
Watery stools, 338
vomit, 328
Waxy casts, 380
Weber's syndrome, 427
Weichselbaum on pus-cocci in urine, 385
Weigert's method of staining tubercle bacilli, 160
Weight, importance of changes in, 31
relation of, to height, 32
Weil, "deep percussion" of intestine, 281
on displacement of border of lungs in expiration, 109
of lower border of lungs in lying down, 109
on dulness in Traube's space in health, 275
on feeling of resistance, 102
on line of relative heart-dulness, 180
on occurrence of thigh-sound, 99
on percussion of spleen, 299, 301
reference to, as improver of percussion, 92
sound over crural artery in mitral stenosis, 22
Wernicke on disturbance of vision from choked disk, 547
on motor aphasia, 507
on sensory aphasia, 506
Wertheim's method of cultivating gonococcus, 549
Westphal on intermitting general paralysis, 485
paradoxical contractions, 486
Westphal's sign, 460
view of tendon-reflexes, 461
Whetstone crystals of uric acid in urine, 387
Whistling râles, significance of, 128
White blood-corpuscles. See *Blood-corpuscles.*
in sputum, 150
Whitish sediment of urine, 363
Whooping-cough, cough in, 143
expiratory bulging in, 88
microbe of, 164
Widal-test for typhoid fever, 247
Widal's reaction for typhoid bacilli, 551
Williams's tracheal sound, 98, 106, 113
Wintrich, reference to, as improver of old percussion, 92
as introducer of percussion-hammer, 93
Wintrich's change of sound, 98, 114
confounded with Williams's tracheal sound, 114
interrupted, 114
Wire pulse, 210
Woillez's cyrtometer for measuring thorax, 140
Wolpe on oxybutyric acid in urine, 407
Word-deafness, 506
testing for, 509
Wounds of the lungs, emphysema from, 50
Writing, center for, 505
diagnostic value of character of, 513
loss of power of, in atactic aphasia, 508
power of, how acquired, 503
Wunderlich's table of temperatures, 55

XANTHIN, concretions of, in urine, 391

YEAST-FUNGUS in feces, 349
in urine, 385
in vomit, 331
Yellow atrophy of liver. See *Atrophy.*
fever, bloody vomit in, 329
skin, 40

ZIEHL-NEELSEN method of staining tubercle bacilli, 161
Ziehl on tract of taste, 421
Zimmerlin on reaction of degeneration in myopathic muscular atrophy, 483
Zygomatic nerve, distribution of, 448

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